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Anesthesiology Supplement
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The Use and Abuse of Airways

FRANCIS J. SMITH, D.O., F.A.O.C.A.
Emeritus Professor of Anesthesiology
Philadelphia College of Osteopathy
Philadelphia

General anesthesia requires the maintenance of an unimpeded airway through which the patient may breathe. An open airway is absolutely necessary to insure satisfactory respiration; it is impossible to overestimate its value during anesthesia.

The patient who breathes poorly during anesthesia is not only poorly anesthetized, but is poorly oxygenated, which is far more serious. One of the common causes of difficult breathing during narcosis is obstruction of the airway above the glottis and, more particularly, obstruction produced by falling of the base of the tongue and the larynx against the posterior wall of the pharynx. This condition is brought about by relaxation of the suprahyoid and infrahyoid muscles which normally act as a cord stretched between the sternum and the lower edge of the jaw and pull upward to the cartilaginous structure of the larynx and hyoid bone, which are fixed midway in this stretched cord. The tongue, inserted in the hyoid and the opening of the larynx, is thus separated from the posterior wall of the pharynx.

Often it is only necessary to properly position the anesthetized patient's head by placing a small pillow under the head and pulling the chin upward. This stretches the tissues between the chin and the sternal notch and pulls the floor of the mouth and base of the tongue forward, away from the posterior pharyngeal wall, thus opening the pharynx. Again, simply turning the head to one side or the other and keeping the jaw forward may insure the patency of the pharyngolaryngeal pathway. In other cases this method is not sufficient to correct the obstruction, and it is necessary to resort to artificial aid. The use of an atraumatic pharyngeal airway, oral or nasal, will frequently suffice. However, in any procedure where the patency of the airway is likely to be threatened by the position of the patient or the nature of the operation, intubation is both advisable and essential.

APPARATUS

Artificial airways are rigid or semirigid tubes or catheters, made of rubber, silk, metal, or plastic, which fit into the upper part of the respiratory tract. They provide a to-and-fro avenue for air, oxygen, and anesthetic gases and simplify the removal of secretions from the pharynx, larynx, and trachea.

There are four types of airways: (1) oropharyngeal, (2) nasopharyngeal, (3) oroendotracheal, and (4) nasoendotracheal.

Oropharyngeal airways are tubes made to conform to the shape of the tongue and the palate. They extend from the lips backward over the dorsum of the tongue to the pharynx and are designed to keep the natural airway open.

There are four kinds of oropharyngeal airways:

1. Those made of flat curved semihard rubber hollow tubes, known as the Guedel, with a flange at the mouth end to fit the lips

2. Those made of metal, similar in design to the rubber airway, known as the Connell

3. Those made of wire, similar in shape to the metal tube, but consisting of a framework of strong wire with the added advantage of less likelihood of obstruction by secretions (They have no flange at the upper end, and may be prevented from falling down into the mouth by threading a strip of gauze bandage through the end. They are known as the Lombard.)

4. Those made of plastic, similar in design to the rubber tube, except that the air or gases pass along the sides instead of through the tube.

All types of oral airways come in various sizes, are introduced in the same way, and serve the same purpose. It is important to select the proper size tube for the individual patient. If it is too small it will not support the relaxed structures. If the tube is too large it extends too far into the hypopharynx and may excite the pharyngeal reflex and produce laryngospasm. In either case, obstruction may occur. Great care must be used with either type of metal tube so as not to traumatize the lips, the teeth, the tongue, or the pharynx. The rubber and the plastic tubes minimize the possibility of trauma. The improperly inserted airway, though of the proper size, may produce obstruction. It is necessary to be sure the flange of the airway is over the patient's lips, otherwise there may be trauma to the lips or gums. The patient should be in plane two of stage three before the oropharyngeal tube is introduced. After insertion, the anesthetist should pull the chin upward and listen to the breathing to be sure the airway is unobstructed.

Nasopharyngeal airways are curved soft rubber or plastic tubes or catheters which extend from the anterior nares to the nasopharynx just below the base of the tongue and serve as an efficient airway as far as the larynx. The tube should be an inch longer than the distance from the tip of the nose to the tragus. The pharyngeal end is beveled laterally and the nasal end is enlarged to prevent it from slipping down the nose. The diameters range from 26 to 32 French. The nasal tube may be inserted under light anesthesia, plane one of stage three, and often may be used when it would be difficult or impossible to introduce an oral airway. The tube should be lubricated with Vaseline or an anesthetic jelly before using. The tip of the nose is elevated and with the curve of the tube pointing forward, the tube is advanced along the floor of the nose close to the septum to avoid injury to the turbinates. Force should not be used, for it may result in trauma and epistaxis. At times it may be necessary to shrink the mucous membrane with a cocaine spray in order to lessen the likelihood of epistaxis. It is seldom impossible to insert a tube in one or the other side of the nose. If the tip of the tube is blocked by the posterior pharyngeal wall, the tube is rotated or withdrawn slightly. New Davol plastic tubes are marked "right" and "left," but because there is an opening opposite the beveled end they may be used interchangeably. After the tube has been placed, it must be ascertained that the patient has a satisfactory air exchange. If there is no enlargement on the nasal end of the tube, as is the case when an endotracheal tube is cut down to make a pharyngeal tube, a safety pin should be inserted transversely and fastened through the end of the tube.

Endotracheal airways are catheters or tubes made

of rubber, silk, flexible metal, or plastic. They extend through the mouth or nose into the pharynx and through the glottic opening into the trachea, thus providing an unobstructed airway. There are two types: the nasal and oral.

Nasoendotracheal catheters are all of the Magill type and are made of soft thin-walled rubber or plastic tubing. They have a beveled tracheal end, similar to the nasopharyngeal tubes. They usually possess the curvature of a circle whose radius is approximately 25 to 30 cm. (10 to 12 inches). They vary in length up to 28 cm. The distance the catheter is introduced may be estimated roughly by placing the catheter along the anterior surface of the neck from the suprasternal notch to the tip of the chin. The lumen of the catheter varies from 28 to 40 French for adults and 18 to 30 French for children, or it may be designated by an arbitrary number from size no. 1 for the infant to size no. 9 for the extra-large patient.

Oroendotracheal catheters are of various kinds:

1. The plain rubber or plastic tube is soft but sufficiently rigid to prevent kinking or collapsing under ordinary conditions.
2. The silk tube is woven and coated with an impervious material which makes it somewhat less than rigid.
3. The so-called Anode tube is made of latex and incorporates a metal spiral in its wall. This metal spiral makes the tube semirigid and prevents kinking or collapse.
4. Lastly there is the Woodbridge tube, made of flexible metal spiral tubing. This type is fitted with a trocar which keeps the tube straight and makes for easy intubation, and when withdrawn allows the flexible tube to conform to the contours of the pharynx and the larynx. To make the tube airtight, a snug Penrose drain is fitted over the outside.

Of all the technical improvements designed to make the administration of general anesthesia simpler, safer, and more efficient, endotracheal intubation is one of the most notable. The adoption of intubation by anesthetists was only gradual; for years this method was used almost wholly by laryngologists. Although the latter often appeared to surround direct laryngoscopy and bronchoscopy with an aura of mystery which undoubtedly had its effect in warding off the anesthetists, it is likely that lack of initiative was an important factor in delaying the entry of the anesthetists into this field.

In 1921, inhalation anesthesia was given "a shot in the arm" through the introduction of blind intubation by Rowbotham and Magill.[1] The new method simplified endotracheal intubation and quickly became routine. This had a stimulating effect on the development of the technic of intubation on the part of anesthesiologists. It was unfortunate that many, carried away by their enthusiasm and their ego, used the new technic in almost every case, often with disastrous results, so for a time the method fell into disfavor in many quarters.

As simple as it may be to the experienced in most cases, intubation, either by the oral or the nasal route, can be bungled, and the patient is the one who suffers. Even when properly performed, the method has certain dangers of sufficient importance to limit its use to cases where it is really necessary or especially indicated. Indications for endotracheal administration are clear-cut and should never be exceeded merely for exhibition purposes.

INDICATIONS FOR ENDOTRACHEAL ANESTHESIA

1. For operations about the head and neck, especially when the usual type of inhaler would obstruct the operative field
2. In any procedure where the position of the patient is apt to threaten the patency of the airway, such as laminectomy or nephrectomy
3. When the patient is prone, as in the case of spinal tumor, Pott's disease, and cervical laminectomy
4. For operations on the brain where the anesthetist is far removed from the patient
5. In cases of respiratory obstruction where the condition cannot be relieved by pharyngeal airways
6. For abdominal operations, especially cholecystectomy, gastrectomy, and for intestinal obstruction, or whenever there is danger of aspiration of fluid regurgitated from the stomach and bowel (Intubation, however, should not exclude the use of gastric lavage. In these cases too, an endotracheal tube protects against the Breuer-Luckhardt reflex which initiates laryngeal spasm.)
7. In operations in which the extreme Trendelenburg position is employed, especially in the obese patient
8. For operations on the thyroid gland (It insures a good airway when pressure from an enlarged gland may threaten collapse of the trachea or offer a marked obstruction to respiration.)
9. For intrathoracic procedures or other types of surgery in which positive pressure is required, an endotracheal tube is imperative.
10. Where controlled or assisted respiration has to be employed, as in the case of curarized patients.

MISUSE AND ABUSE OF THE ENDOTRACHEAL TUBE

Technical mistakes on the part of the anesthetist may lead to the development of numerous complications. Bleeding readily occurs from an inflamed and engorged nasal mucosa when blind intubation is attempted and adhesions of the septum may form later and cause obstruction. Enlarged adenoids may block the passage of a tube through the nose, and their removal in this unorthodox manner may result in considerable bleeding. Septic material from infected nasal sinuses may be carried into the trachea by the nasal route.

Mechanical injury due to lack of skill, hurry, impatience, ignorance of anatomy, or excessive force has led to various sequelae. Too large endotracheal tubes which require forceful passage stretch or tear the vocal cords. Continued pressure of large tubes or overinflated cuffs may lead to tracheitis, edema, or even sloughing of the tissues. Tubes that can be passed easily and that will not produce irritation regardless of the length of operation should be used.

The vocal cords are easily damaged and formation of granulomas has been reported frequently. This complication may occur even when intubation is performed under direct visualization but is more common when the blind technic is used. The lesion is usually found at the posterior end of a vocal cord or on an arytenoid cartilage. It may be bilateral and follow a hematoma or an abrasion produced during manipulation of a tube past the vocal cords. It may be due to sustained pressure from an S-shaped distortion of a tube which persists when the patient's head remains in extension throughout the operation. A plastic tube which becomes softer at body temperature is less likely to cause such damage.

Intubation during a light plane of anesthesia may cause laryngospasm. If this occurs before the tube is passed, considerable force is necessary to insert the tube, and if it occurs after intubation, the glottis will close down on the tube and coughing and bucking follow. Such spasmodic movements cause grinding of the tube against the cords and the mucosa of the trachea, and much trauma may result. Similar damage may occur at the end of the operation if the patient is allowed to waken while the endotracheal tube is still in place. Spasm and coughing at such a time may well prove dangerous.

Hyperextension of the head and neck angulates the trachea at the larynx and renders intubation more difficult. In order to overcome improper positioning of the head, excessive manipulation of the blade of the laryngoscope is necessary. By raising the head on a small pillow, 4 or 5 inches above the table, and then extending the head on the neck, the passageway is brought from the incisor teeth to the larynx and trachea, normally a right angle, into a straight line. This so-called sniffing position greatly facilitates laryngoscopy.

Movement of the endotracheal tube during anesthesia may result in considerable irritation or trauma to the vocal cords and the mucosa of the trachea. This could be the result of a heavy to-and-fro soda lime canister dragging on the tube. A gasping or tugging type of respiration which saws the tube back and forth across the cords is another cause of movement of the tube.

The type of tube used is important, the conventional rubber one with fixed convexity being more likely to cause trouble than one made of vinyl portex (plastic) which becomes appreciably softer at body temperature and conforms better to the shape of the pharynx and trachea. Trauma associated with pressure of a tracheal tube or pack is likely to be aggravated by exceptional dryness of the air passages. This dryness results from the use of atropine or scopolamine or by drying of the mucosa by the continuous flow of oxygen in nonrebreathing or insufflation types of anesthesia, but particularly because the tube by-passes the nose, nature's air-conditioner.

Perhaps the greatest neglect in the technic of intubation has been in the matter of cleanliness. Soiled endotracheal tubes may carry a wide variety of organisms into the trachea and rub them against the delicate membranes for hours on end. The anesthetist who lacerates the glottis and then rubs a dirty tube across the wound has committed a serious offense. Contamination certainly must be considered a real hazard in endotracheal intubation.

Every type of airway should be cleaned as soon as possible after use. First it should be washed with soap and warm water and the lumen thoroughly cleaned with a long flexible wire-handled brush made for the purpose, or, in the case of oropharyngeal airways such as the Guedel or the Connell, a strip of gauze wet with soapy water attached to a wire can be run back and forth through the lumen. It is then rinsed thoroughly in plain warm water to remove all traces of soap. Then it is placed in 70 per cent alcohol or aqueous Zephiran for 30 minutes. Metal tubes may be boiled for 10 minutes, but boiling causes deterioration of rubber and plastic tubes. Lastly, the tube is again rinsed thoroughly in warm water, dried, and stored in a drawer or cabinet where it will not be exposed to air currents, handling, or contact with unclean objects.

Before concluding this paper it is necessary to mention some of the more serious conditions that may be associated with intubation. Mechanical or chemical irritation of the respiratory system may produce cardiac arrhythmia, ventricular fibrillation, or even cardiac arrest by reflex action. These reflexes may originate in the oropharynx, nasopharynx, larynx, or trachea, and it is these that are of concern. There are a number of reports of cardiac arrest or sudden cardiovascular failure possible due to serious reflex cardiac derangements during the introduction of pharyngeal airways, both oral and nasal, during endotracheal intubation or extubation, or during endotracheal suctioning, all under anesthesia. Waters and Gillespie[2] reported one death on intubation and one death on placing of an oral pharyngeal airway under cyclopropane anesthesia. Nosworthy[3] reported two similar cases under cyclopropane. Nelson[4] reported fatal cardiac arrest under cyclopropane on intubation before surgery. Dripps and his associates[5] reported a case of arrest probably due to a vagovagal reflex, secondary to intubation during cyclopropane anesthesia. Ament, Papper, and Rovenstine[6] reported two cases of arrest probably due to inflation of an endotracheal cuff during cyclopropane anesthesia. Gillespie[7] told of a fatality on intubation under light nitrous oxide-oxygen-ether anesthesia. Smith and Nolan[8] have cited a case of cardiac arrest on intubation under Pentothal-curare. Dwyer[9] reported a similar case. Bishop[10] and Rovenstine[11] each tell of a case of death under intubation. Dale[12] reported five cases of cardiac arrest with various anesthetics as intubation was being performed, indicating reflex cardiac inhibition superimposed upon probable hypoxia.

Scurr[13] has reported a case of cardiac arrest thought to be the result of inflating the endotracheal cuff. Bishop[10] cited death in a child on intubation under ether. Johnstone[14] reported a fatality believed to be due to a vagovagal reflex, initiated by respiratory obstruction and difficulty in intubation. Shumacker and Hampton[15] reported four cases of arrest during endotracheal suctioning and two cases during extubation under ether. Wylie[16] reported a case of arrest in an awake patient during endotracheal suctioning. Unquestionably there have been many, many instances of such accidents that have not been reported or have escaped my attention.

The relationship between hypoxia, asphyxia, and reflex cardiac inhibition is still under investigation. However, practical experience indicates that hypoxia and, especially, asphyxia increase the cardioinhibitory effect of vagal stimulation. Another important factor in the development of reflex cardiac disturbances is the depth of anesthesia. King and his coworkers[17] state that, in the absence of hypoxia, reflex cardiovascular disturbances during laryngoscopy and intubation depend primarily upon the depth of anesthesia. Second plane, stage three anesthesia is necessary for easy, successful, and safe endotracheal intubation.

SUMMARY

The various kinds of airways have been discussed. Artificial airways are of great technical aid to the anesthetist in keeping an open airway at all times. Of these aids the endotracheal tube or catheter is the greatest aid since it guarantees the patency of the airway, not only in the mouth and pharynx but in the larynx and trachea as well.

Indications for the use of airways have been reviewed.

Various sequelae, minor and major, have been cited. When an airway is to be used, careful discrimination as to the type, the technic to be employed, and meticulous management must be constant safeguards in its application.

6445 Greene St.

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Barbiturate Hypnosis in Pediatric Anesthesia

LAWRENCE EVERETT GIFFEN, D.O.
Jefferson City, Mo.

A stay in the hospital, even though for a relatively simple procedure such as a tonsillectomy, is one of the most distressing and disturbing periods a youngster may experience.

Proper psychologic preparation of the child, by both the parents and family physician, is a valuable aid and does much to eliminate the fear of the unknown in the child. However, many of us still remember rather vividly the time when we, as children, entered the hospital. Even though we were only 5 or 6, we may remember fighting or struggling when ether was administered. The psychic trauma resulting from the surgical procedure or hospital stay at that early age may result in a fear of doctors or hospitals in later life.[1]

Telling a child that he will be all right and that he has nothing to fear is not enough, especially when he is taken from his mother's arms, transported through unfamiliar surroundings, and placed on a hard operating room table. Strange people in white then put towels over his eyes and face and begin to administer the anesthetic. Most children, under those circumstances, will be extremely apprehensive, frightened, and struggle to get away.

With this in mind, and with the hope of making the hospital stay a pleasant experience for the small child, it was decided to learn if barbiturate hypnosis induced before anesthesia would lessen the psychic trauma of operations on children.

METHOD

This report is based on ninety-six pediatric operations in which the children received a barbituric acid derivative. Sixty-four children were given Surital and thirty-two Pentothal.* These drugs were administered rectally by the anesthesiologist while the child was still in his room and with one or both parents present. After the child was asleep, he was transported to the surgical suite for administration of the anesthetic and surgery.

The Surital and the Pentothal were freshly prepared for each patient. The 0.5-gram or 1-gram ampule was dissolved in tap water to make a 10 per cent solution. Following the method of Migdal, Goldfeder, and Kornblau,[2] the dose used was 15 mg. per pound of body weight. In no case was the dose greater than 1,000 mg., or 1 gram, regardless of the patient's weight.

The anesthesiologist administered the drugs from a 10-cc. syringe through a small urethral catheter. The only premedication given was atropine sulfate, in the dosage recommended by Leigh and Belton,[3] 45 minutes to 1 hour before surgery. Open drop ether was the anesthetic in all of the cases.

In the Surital series the age distribution ranged from 4 weeks to 17 years. The average age was 7.2 years. There were 30 boys and 34 girls. The weight range of the children was 7.5 to 145 pounds. Their average weight was 57.7 pounds.

Of the group receiving Pentothal, there were 21 boys and 11 girls. Their ages ranged from 2 months to 14 years. The average age of this group was 6.1 years. Their weight ranged from 11 to 112 pounds, and the average weight was 57.5 pounds.

Of the children receiving Surital, seven did not go completely to sleep after having been given the drug. However, six were drowsy and were not aware of their surroundings. The average time was 13.3 minutes between administration of Surital and the time of sleep.

All but one child who received Pentothal were asleep, and that one was drowsy and not aware of his surroundings. The average time, following administration of Pentothal until the time of sleep, was 10.4 minutes in the remaining 31 cases.

The average length of surgery in the Surital series was 21 minutes, and in the Pentothal series it was 21.8 minutes. This is a relatively short operating time; however, the majority of operations were tonsillectomies.

In the Surital series the time from surgery to response averaged 69 minutes. In the Pentothal series it averaged 59.9 minutes.

After awakening, most of the patients fell asleep again and slept for several more hours. This was considered advantageous, particularly in tonsillectomies, since the children required no further sedatives or drugs for pain relief.

*Pentothal was supplied through the courtesy of Abbott Laboratories.

TABLE I—AGES OF CHILDREN RECEIVING SURITAL

Age in Years	Number
1	3
2	1
3	2
4	2
5	12
6	11
7	4
8	8
9	3
10	9
11	3
12	1
13	3
14	0
15	1
16	0
17	1
Total	64

TABLE II—OPERATIVE PROCEDURE FOR CHILDREN RECEIVING SURITAL

Tonsillectomy	48
Appendectomy	9
Hydrocele	2
Congenital pyloric stenosis	1
Hernia	1
Fracture	1
Tendoplasty	1
Circumcision	1
Total	64

TABLE III—AGES OF CHILDREN RECEIVING PENTOTHAL

Age in Years	Number
1	1
2	1
3	1
4	1
5	3
6	9
7	3
8	3
9	1
10	4
11	2
12	1
13	1
14	1
Total	32

TABLE IV—OPERATIVE PROCEDURE FOR CHILDREN RECEIVING PENTOTHAL

Tonsillectomy	21
Appendectomy	6
Skin graft	1
Dilatation of lacrimal duct	1
Repair of head laceration	2
Open reduction	1
Total	32

RESULTS

Fifty-six children in the Surital series and twenty-eight in the Pentothal series were 5 years of age or older. These children were questioned on the day following surgery regarding their memories of the previous day. The result of the questions are shown in Table V.

Some of the children, who had been asleep in their rooms but answered that they remembered going to surgery, did rouse slightly while being transported to the cart; however, for the most part they went back to sleep immediately.

TABLE V—CHILDREN'S ANSWERS TO QUESTIONS

Question	Surital Hypnosis		Pentothal Hypnosis	
	Yes	No	Yes	No
Was it a pleasant experience?	49(87.5%)	7	23(82.1%)	5
Do you remember going to surgery?	16(28.5%)	40	5(17.8%)	23
Do you remember getting anesthetic?	12(21.4%)	44	6(21.4%)	22
Would you come back to the hospital?	55(98.2%)	1	28(100%)	0

REMARKS

The incidence of nausea and vomiting following this type of anesthesia was considerably less than is usual in such cases. While the number was not statistically recorded, the surgeons noted and remarked about this during their postoperative care of the patients.

All but one of the children took the anesthetic satisfactorily. In this case, the child did not receive the usual effect from Surital and was very restless during the induction of ether anesthesia. He was extremely restless following surgery and, before and after waking, required constant attendance. No explanation for this untoward behavior can be given other than that he may have been sensitive to thiobarbiturates.

It was noted in both series of cases that the depth of respiration was more shallow than is usually noted in children during ether anesthesia following the usual premedication. Vigilance is required so that the patient does not slip into too deep a plane of third stage anesthesia.

No attempt was made to measure the amount of ether used, but it is believed that about one-half as much was used with barbiturate hypnosis as with other types of premedication. In many cases, after the patient was relaxed enough to admit the mouth gag, surgery was completed without ether insufflation.

SUMMARY

A clinical evaluation has been made of 96 pediatric operations in which barbiturate hypnosis was induced before anesthesia.

The findings are summarized as follows:

1. Psychic trauma was reduced.
2. Uncooperative children were more easily handled.
3. There was less screaming, kicking, and thrashing about by the small patient in the operating room.
4. Induction of ether anesthesia was smoother than usual. It is believed that less ether was used than in operations in which barbiturate was not used.
5. The patient required less analgesia following surgery. Nearly all of the patients were drowsy most of the day of surgery.
6. Of the children old enough to be questioned, 98.8 per cent indicated that the hospital stay was pleasant and that they would not mind coming back in the future, if necessary.
7. There was no significant clinical difference between Pentothal and Surital as a hypnotic drug in rectal anesthesia.

209 Monroe St.

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Clinical Evaluation of Inhalation Technics in Pediatric Anesthesia

BORIS H. TRAVEN, A.B., D.O.
Woodbury, N.J.

Increasing recognition and knowledge of the special problems of anatomy and physiology of infants and children have brought with them rapid advances and refinements in pediatric anesthesiology. Whereas surgery has been divided into major and minor categories, every anesthetic must be considered a major procedure in which careful evaluation and eternal vigilance are necessary for a satisfactory result.

It has been said that good anesthesia means minimal disturbance to basic physiology; it implies utilization of methods and combinations of agents without interference in the metabolic functions of vital organs, adequate but not overabundant oxygenation associated with proper elimination of carbon dioxide, maintenance of cardiac dynamics, and the preservation of electrolytic balance in each individual.[1] This paper will present some of the factors pertinent to the anesthetization of pediatric patients and evaluate the technics employed by the Department of Anesthesiology of the Metropolitan Hospital, Philadelphia. With these objectives in mind a consideration of the special problems of infants and children is of utmost importance.

The small size of the anatomic structures, the encroachment on the air space of tonsils and adenoids which are frequently hypertrophic, the incomplete development of the bony thorax, the relative immaturity of the intercostal muscles, the relative instability of the cardiovascular system, and the incomplete development of the central nervous system must be reckoned with in providing a safe and satisfactory anesthetic for the infant and child. These anatomic factors assume much greater significance, however, in the way in which they modify the physiologic functions of the pediatric patient.

According to Deming[2] maintenance of normal respiration and circulation during induction of anesthesia is a major factor in the subsequent maintenance of anesthesia and in the general well-being of the child. Although circulatory fluctuations are greater in infants than in adults, cardiovascular depression responds most readily to a few minutes of adequate oxygenation and ventilation. Despite the wide variations in normal circulatory response, infants and children seem less susceptible to circulatory accidents than do adults.

Observation of respiratory activity of the infant and young child reveals it to be rapid in rate and regular, with an occasional deep breath. In children under the age of 9, the tidal exchange is 50 to 250 cc. with an average of 150 to 200 cc.[3] Oxygen consumption varies from 7 cc. per kilogram of body weight per minute, almost twice that of the adult, to a peak of 10 cc. per kilogram of body weight per minute at 3 to 5 years of age.[2] Since approximately one-third of the tidal volume represents dead space, any aberration of respiratory activity may produce peripheral and central exhaustion and inadequate gaseous exchange. Griggs, Adriani, and Berson[3] state that it is not the size of the space one breathes from which is important but the size of the space one rebreathes from which should receive attention. The dead space should be abbreviated. Respiratory difficulties are more frequent in pediatric anesthesia than are severe circulatory disturbances. Hypoxia secondary to respiratory complications is fairly well tolerated for short periods, but clinical evidence of central nervous system damage will be present if it persists for longer than 3 or 4 minutes. Severe circulatory disturbance with ischemia in addition to hypoxia produces irreparable damage in a matter of seconds.[2] Indeed, although children do succumb from overdosage of anesthetic agent, the commonest cause of mortality in the operating room is anoxia.[4]

The immaturity of the infant's central nervous system accounts for the minimal function of the cortical and some subcortical centers. Accordingly, the stages and planes of anesthesia are ill defined and difficult to interpret in infants as compared to older children and adults.

Acidosis requires correction preoperatively when it exists. Metabolic acidosis is often present in toxic infants and children and should be corrected by adequate fluid replacement, et cetera, prior to induction of anesthesia. During anesthesia, precautions must be taken to prevent carbon dioxide retention secondary to respiratory depression (anesthetic or obstructive).

This study includes 976 cases enumerated according to the anesthetic technic and agents used (Table I). In this series, which covers a period of 20 months, all types of pediatric surgery were performed, and in every instance anesthesia was produced by inhalation technics.

Except in surgical emergencies, all patients were evaluated preoperatively with a complete history and physical examination, complete blood count, bleeding and coagulation time, urinalysis, and a chest survey x-ray. A 2-quart saline and glucose rectal irrigation was ordered for the night before surgery in order to hydrate the patient and to counteract acidosis.[5]

Premedication was ordered according to the recommendations of Leigh and Belton,[6] and more recently Demerol was ordered in dosage of approximately 1 mg. per pound of body weight.[7] Atropine or scopolamine was given concurrently in appropriate dosage. This premedication was administered 1 hour before surgery, and it has served satisfactorily with no preoperative respiratory depression and minimal apprehensiveness on the part of the patient.

As will be noted in the data presented, many variations in technic were employed and demonstrated. During the first 6 months of this series, endotracheal technics were rarely used. Consequently, ether-air insufflation was used in maintaining anesthesia for tonsillectomy and adenoidectomy or the open drop ether technic was used for maintenance in other procedures. These technics left much to be desired because of the difficulties experienced in maintaining an adequate airway, the constant danger of aspiration of mucus and blood, the imminence of hypoxia because of these, and frequent fluctuation of the level of anesthesia.

Inevitable partial anesthetization of the surgeon, his assistants, the anesthesiologist, and the other operating room personnel did not contribute to the tranquillity and understanding of each other's problems so necessary for efficiency in the operating room. In those cases where the nitrous oxide-oxygen-ether sequence (carbon dioxide absorption by circle filter) was employed for induction and maintenance, the disadvan-

tages of this technic were observed with alarming frequency. Dead space could not be reduced effectively without the insertion of new mushroom valves into the anesthesia machine, a new type of chimney piece, new masks and the like.[3] Carbon dioxide retention, rebreathing, and valve resistance could not be coped with satisfactorily. Accordingly, a re-evaluation was made of the technics in pediatric anesthesia and the methods currently employed are described.

Induction is performed by the use of either the nitrous oxide-oxygen-ether sequence by the semiclosed technic described by Stein[5] in older children, or by the open drop Vinethene-ether sequence in infants and young children. A few drops of essence of peppermint on the gauze of the ether mask facilitates a smoother, more pleasant, and more rapid induction in most instances. Direct oxygen is administered under the ether mask whenever necessary and is always used for infants to provide adequate oxygenation, to reduce rebreathing to a minimum, and to prevent gaseous acidosis.[1,2]

Preparations are made for intubation as soon as the jaw is relaxed.[8,9] The mouth, oropharynx, and laryngopharynx are carefully aspirated to remove accumulated secretions. The head is then elevated slightly and extended. A small pillow placed under the occiput will facilitate this positioning of the head. The laryngoscope is then gently inserted into the mouth, the tongue and epiglottis are lifted upward and slightly forward, and the larynx and vocal cords are directly visualized. (In most instances a medium sized Wis-Foregger laryngoscope blade mounted on a medium handle serves satisfactorily. Rarely, a small blade is used.) A Portex plastic tube (Magill) of appropriate diameter fitted with an adapter is selected, lubricated with a small amount of fat-free water-soluble lubricant containing a topical anesthetic,[5] and passed gently into the trachea. It is connected to a nonrebreathing valve which is in series with a latex rubber gas reservoir of 1.5-liter capacity and with the anesthetic supply tubing of the anesthesia machine. With this arrangement, the plane of anesthesia may be accurately controlled in young children by the appropriate admixture of nitrous oxide, oxygen, and ether. In children over 65 pounds in weight, the nitrous oxide-oxygen-ether endotracheal technic with carbon dioxide absorption by circle filter is more effective than the nonrebreathing method for maintenance of anestheisa. If the operation is not in the region of the head or neck, an oropharyngeal airway is inserted into the mouth as a bite block. A mouth gag serves in its stead if tonsillectomy and adenoidectomy are to be performed.

At the end of the operative procedure the oropharynx and laryngopharynx are again aspirated, and tracheal aspiration is carried out whenever necessary. Extubation is then done, and the patient's head is positioned, airway inserted, and aspiration is repeated to assure a patent airway. In many instances patients have reacted shortly after extubation while the patient was still in the operating room. In all cases, reaction was complete soon after patients had been returned to their rooms. Positioning of the patient on the carrier for postoperative return to his floor as well as when placed in bed has been standardized in our hospital.[5]

TABLE I*

Anesthetic and Technic	Number
Vinethene open drop	13
Ethyl chloride-ether open drop	105†
Vinethene-ether open drop	202†
Ether open drop with direct oxygen	6
Nitrous oxide-oxygen-ether sequence with ether-air insufflation	47
Nitrous oxide-oxygen-ether sequence with circle filter carbon dioxide absorption	42
Nitrous oxide-oxygen-ether sequence with to-and-fro carbon dioxide absorption	2
Vinethene-ether open drop, nitrous oxide-oxygen-ether to-and-fro absorption	3
Nitrous oxide-oxygen-ether endotracheal to-and-fro absorption	4
Nitrous oxide-oxygen-ether endotracheal circle filter absorption	23
Nitrous oxide-oxygen-ether endotracheal non-rebreathing	36
Nitrous oxide-oxygen-ether, ether-oxygen endotracheal (Flagg technic)	1
Vinethene-ether, ether-oxygen endotracheal (Flagg technic)	2††
Vinethene-ether open drop, nitrous oxide-oxygen-ether endotracheal (Ayre's tube)	1
Vinethene-ether open drop, ether-oxygen endotracheal nonrebreathing	98
Vinethene-ether open drop, nitrous oxide-oxygen-ether endotracheal circle absorption	6
Vinethene-ether open drop, nitrous oxide-oxygen-ether endotracheal nonrebreathing technic	372
Ethyl chloride-ether open drop, nitrous oxide-oxygen-ether endotracheal nonrebreathing technic	6
Ethyl chloride-ether open drop, ether-oxygen endotracheal to-and-fro absorption technic	7

*In addition to the anesthetics administered by the author, those of M. L. Stein, D.O., and M. B. Levine, D.O., have also been included in these totals for the purpose of completeness. Their cooperation in this study is deeply appreciated.

†Includes anesthetics administered with and without subsequent ether-air insufflation.

††Oxygen or air used in this technic.

COMMENTS

With few and rare exceptions the endotracheal technics have been received with enthusiastic approval by the departments of surgery and anesthesiology. Even in instances in which the surgeon can perform the operation in 15 minutes or less, this technic is the method of choice for the operator as well as for the anesthesiologist. Leigh and Belton[6] and Thomas[8] point out the advantages of endotracheal anesthesia and list them as follows: (1) freedom of airway, (2) reduction of dead space, (3) control of pulmonary ventilation, (4) removal of the anesthesiologist from the immediate operative field, and (5) reduction of the amount of anesthetic used.

It has been my experience with endotracheal technics that all the advantages have been most satisfactorily fulfilled. I believe the lighter plane of anesthesia as well as the appreciably smaller total dosage of anesthetic agents used is responsible for the optimal result of patients reacting while still in the operating room or shortly after return to their room. The free airway, minimal dead space, and the ease of control for ventilation as well as for assisted respirations, even when the anesthetist is removed from the operative field, have been additional incentives for the ever-increasing use of endotracheal technics.

Disadvantages have played little or no part in the over-all picture.[8] There have been a few instances of traumatic removal of loosened deciduous teeth, but oral or laryngeal trauma or lacerations have not occurred. I recommend the removal of loosened deciduous teeth as a precaution against their accidental aspiration during the postoperative period. Extubation laryngospasm has been minimized by the use of an anesthetic lubri-

cant on the tube. With the cooperation of the department of otorhinolaryngology, procedures have been modified so that the presence of an endotracheal tube in the patient's mouth does not interfere with the surgeon.

Portex plastic tubes (Magill), which we now use in pediatric anesthesia, have certain advantages worthy of mention. When placed in cold sterilizing solution they assume a stiffness which facilitates intubation. Once the tube is in position, the patient's body temperature softens the tube sufficiently to make it conform to the shape of the structures surrounding it, thus minimizing trauma due to pressure. There is always sufficient stiffness to preclude kinking or obstruction due to external pressure of the tongue blade, sponge, or other instruments when tonsillectomy and adenoidectomy are performed.

COMPLICATIONS

In addition to the traumatic removal of loosened deciduous teeth, occasional instances of tracheitis have occurred. Increased precautions in sterilization of the plastic tubes should reduce the incidence of this complication. One child in the series developed postoperative laryngeal edema which responded promptly to aspiration and steam inhalations, and he was discharged in 48 hours. One child expired suddenly approximately 20 hours postoperatively after a tonsillectomy and adenoidectomy under Vinethene-ether open drop induction and nitrous oxide-oxygen-ether orotracheal nonrebreathing technic. Coroner's autopsy revealed normal oral, laryngeal, tracheal, and bronchopulmonary structures, and cause of death was attributed to air embolism in the superior vena cava. It is difficult to explain this fatality since there were no open sinuses in the tonsillar or adenoid fossae, and no intravenous injections had been given.

SUMMARY

1. Some pertinent facts relative to the anatomy and physiology of the cardiovascular, central nervous, and respiratory systems of infants and children have been discussed.

2. A report has been rendered on a series of 976 anesthetics administered to pediatric patients for all types of surgery at the Metropolitan Hospital. They have been subdivided according to the agents and inhalation technics used.

3. The present methods of choice at the Metropolitan Hospital have been described in detail. Advantages, disadvantages, and complications have been noted and discussed.

102 Delaware St.

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Anesthesia in the Small Hospital

PAUL A. STERN, B.S., D.O.
Dallas, Tex.

INTRODUCTION

Today, most anesthesiologists receive their formal training in one of the many larger osteopathic hospitals in the country. In these teaching centers they experience a type of anesthesia practice, characterized by an abundance of equipment, supplies, help, leisure time, diversity of cases, and compensation. These teaching conditions can make for a disenchanting experience, when, with certificate in hand, high hopes and ideals, the recently graduated resident sets out to take up his position in a small osteopathic hospital. Here he will find none of the luxurious facilities that made for pleasant working conditions, and that all the responsibilities of his department have fallen upon him. However, if his training has been adequate, and his imagination remains intact after his residency, he will be able to render a service to his hospital, the patients, and the staff that is equal, and often superior, to the quality of that seen in the teaching center from which he recently departed.

On arrival, the anesthesiologist will often find in the small hospital an anesthesia apparatus of dubious vintage. If this instrument is not approached with prejudice, he will find that it can be made serviceable with a general cleaning and overhauling.

After providing for the source of anesthetic gas and oxygen, the anesthesiologist will often find that the accessories necessary for its application are absent or not available in sufficient quantity. Additional face masks are needed, endotracheal catheters must be had in a variety of sizes, and connectors and slip joints are essential. The business director of the hospital may find the list of new accessories and other pertinent equipment staggering, even though the need for the equipment is urgent or basic.

However, the confidence and cooperation of the business director of the hospital may often be won if the anesthesiologist displays thrift and ingenuity by improvising new equipment from items commonly available in any hospital. One such improvisation of great general benefit is made from a breast pump from the obstetrics department. With slight alteration the pump can be converted to a bedside suction unit to be used after patients have returned from surgery, especially tonsillectomy. Attaching a suction bottle and a piece of rubber tubing with a polyethylene suction catheter is not only a money-saving procedure, but often a life-saving one. Many of the dangers of a potentially hazardous immediate postoperative period have been minimized by having such a suction unit at the bedside.

Similarly, for tonsillectomy it is not difficult, with a supply of rubber tubing and a few Y-connectors, to

convert the common ether insufflator for use in a modification of Ayre's technic.[1] With rubber tubing and a wash bottle, a source of oxygen for endotracheal insufflation of newborns can be made, using the wash bottle as a safety valve.

RELATIONS WITH STAFF

The next step in the anesthesiologist's progress is to make the surgeons and other physicians on the staff feel that he is an asset to the hospital. A sympathetic regard for the surgeon and his problems is likely to evoke a similar degree of cooperation which will benefit all concerned. Until the anesthesiologist learns the surgical technics preferred by each surgeon, he should have presurgical consultation so that the proper anesthetic technic and agent can be selected. For example, in rectal surgery there are several different accepted positions, the lithotomy, lateral or semiprone, and Buie being the commonest. A patient in the semiprone or Buie position will not do well with inhalation or intravenous anesthesia, because the posture is not conducive to adequate respiratory excursion. If the surgeon has been performing rectal operations in one of these positions with an inhalation or, more commonly, an intravenous anesthesia, it behooves the anesthesiologist to convert him to some form of conduction anesthesia. One of the most convincing ways to accomplish this is to remind the surgeon of the rectocardiac reflex and its inherent dangers and of the other vagal reflexes that can produce cardiac arrest.[2]

While cardiac arrest is under discussion, I wish to point out that the anesthesiologist should prepare the entire staff, and himself, for such an emergency. In surgery he can provide a cardiac arrest kit. At other stations in the hospital a similar kit, consisting of two syringes, a spinal needle, some plain needles, an oropharyngeal airway, some ampules of epinephrine 1:1000, procaine 1 per cent, and a source of oxygen can save the life of a bed patient. In surgery a rib cutter, defibrillator, and a source of endotracheal oxygen should be on hand.[3] The institution of intrathoracic cardiac resuscitation is usually initiated at the order of the anesthesiologist. If each member of the surgical team is aware of his function a rapid and efficient procedure, devoid of confusion, will produce the desired result in many cases.

The practice of conduction anesthesia for obstetrics will earn the appreciation of patients but it will make serious inroads on the anesthesiologist's leisure time. Before he embarks on this field of practice he must have knowledge of proper obstetric practice, equivalent in many respects to that of the obstetric specialist.

In obstetrics all decisions concerning the proper conduct of the anesthesia during labor and delivery will depend on the anesthesiologist. He will be expected to decide which method will be best, what time it should begin, what agent should be used, and whether the anesthesia will delay the progress of labor. Also the use of premedication agents will fall into his domain as well as the resuscitation of the newborn, using endotracheal apparatus.

The anesthesiologist can also advise the obstetrician on the proper choice of inhalation agent and technic and assist him in the premedication of patients as well as the application of pudendal nerve blocks and perineal infiltrations.

The department of internal medicine can offer a great degree of cooperation if the anesthesiologist realizes that a presurgical evaluation by an internist in all poor risk patients will benefit the patient and make his own job during surgery much easier. This presupposes the fact that the internist is aware of the existence of various types of anesthesia, that he can evaluate the cardiac and respiratory reserve, that he can estimate the degree of hydration of the patient, and that he realizes the extent of the surgical insult that the poor risk patient will have to endure. In our own hospital, we feel so strongly about the value of the internist's consultation prior to surgery, that all patients over 50 years must be evaluated before major elective surgery. The routine examination of these patients has unearthed more pathology unrelated to the anticipated surgery than all the routine presurgical chest x-rays that are taken on all patients. In the absence of a qualified internist, it becomes the problem of the anesthesiologist, with or without the help of the surgeon, to evaluate these patients.

In return for the valuable assistance of the internist, the anesthesiologist can help the medical department by performing diagnostic and therapeutic nerve blocks. There is nothing more gratifying to all concerned than the dramatic effect produced by a stellate ganglion block to relieve cerebral arterial spasm, the comfort derived from a Gasserian ganglion block for tic douloureux, or the rapid return to normal renal function when treating a patient with eclampsia or a lower nephron nephrosis with fractional epidural anesthesia.

After having demonstrated his cooperative attitude to the specialty departments on the staff, the new anesthesiologist should make it clear to all physicians, especially the general practitioners, that the choice of anesthetic technic and agent should be left to his discretion. They should be impressed that it is not fair to patients or to the anesthesiologist to make commitments to them without first conferring with him.

The anesthesiologist must also stress the fact that modern practice dictates than an anesthetic should not be given for elective surgery unless the patient has had the benefit of a thorough preparation, including blood count, blood chemistry, urinalysis, chest x-ray, electrocardiogram when indicated, history and physical examination. At the same time it is well to impress the staff that elective surgery must be postponed in patients suffering from anemia, kidney disease, diabetes, and other common pathological conditions until correction has been attempted.

INSTRUCTION OF INTERNS

Another group of doctors on the hospital staff that requires consideration is the intern staff. These doctors expect a certain degree of anesthesia instruction, and it is useful for the anesthesiologist to supply them with training so that they can be of intelligent help to the patient and the anesthesiologist.

At our hospital we begin the intern year with a series of lectures. The first things that we stress are the findings they must elicit during the history and physical examination. We require that history and physical examination include all the necessary vital statistics, height, weight, blood pressure, evaluation of the respiratory and cardiovascular systems, previous surgery, and anesthesia. We emphasize that all abnormalities that may influence the choice or conduct of an anesthesia should be given special written attention on the history sheet. We also stress the importance of being alert for coincident medication which may alter the anesthesia, such as cortisone, ACTH, and disulfiram.

Following these preliminaries the interns receive instruction in the principles of inhalation anesthesia, prevention and treatment of shock, and resuscitation, especially of the newborn. Universally, these doctors are unaware of the practical aspects involved in these problems. They do not know the difference to the patient or to the anesthesiologist in the use of open drop inhalation anesthesia or the semi-open, semi-closed, and closed technics. They are unaware of the pharmacologic effects of the different vasopressors, analeptics, sedatives, and other drugs used by the anesthesiologist. They have little or no ability to pierce a vein, and the use of an 18-gauge needle is a novel experience. The idea of cutting down on a vein or administering parenteral fluid through an artery is new to many interns.

We assign to all of these men a relief role. Frequently the lone anesthesiologist is needed in two places at once. If two relief interns, each with some basic training, are available, the situation can be managed with a reasonable degree of safety to the patient. In return for this assistance by the intern staff, anesthesia is taught to them according to their interest and ability. However, few interns ever feel that open drop ether is an important type of anesthesia or appreciate the knowledge prerequisite for doing spinal blocks, although they feel that it is their inalienable privilege to pierce patients with needles, especially in the back!

ROLE OF NURSES

The last group of professional associates in the hospital is the nursing staff, which falls into two groups, floor nurses and surgery nurses. Nurses can be of great help or hindrance to the anesthesiologist, depending on their training and their aptitude. It is necessary to insist, directly or through the supervisor of nurses, that the floor nurses prepare patients properly for surgery. This means that they must not feed patients prior to surgery, that all dental or other prostheses must be removed, as well as the chewing gum, that they must not allow smoking prior to surgery, that the schedule for administration of preanesthesia drugs must be followed, and that changes must not be made in any orders except at the direction of the anesthesiologist. Nurses can benefit all concerned if they will frequently check patients postoperatively. The early detection of hypotension, dyspnea, apnea, or hemorrhage by a floor nurse can be lifesaving.

In order to achieve this proper preparation of patients it is necessary that the anesthesiologist repeatedly bring it to the nurse's attention, because if he looks around him every 6 or 8 weeks he will notice that some of the nurses on the floor were not there the last time that he looked. In order that new nurses receive instruction the anesthesiologist must be repetitious, consistently redeclaring his rules and regulations.

In the small hospital the surgical nurses usually have the responsibility for preparation and maintenance of the anesthesiologist's equipment. Like the floor nurses they are likely to change frequently, or if they are more constant in their attendance, they tend with time to become lax in their endeavors. These nurses can be of important assistance to the anesthesiologist, especially in case of emergency, if they know where all his equipment is kept so that it can be reached instantly. They must be reminded often to replace worn and defective equipment, such as needles, syringes, and tubing; and it should be pointed out always that the use of such items is dangerous to the patient. The safety of the patient is as much the nurses' responsibility as it is that of any of the physicians on the surgical team, and it is necessary for the anesthesiologist to keep them constantly aware of this fact.

RELATIONSHIP WITH PATIENTS

The patients constitute the final group with whom the new anesthesiologist must deal. Patients in different parts of the country react differently to the idea of anesthesia. On both coasts and in the larger northern cities they tend to show no antipathy toward spinal or other conduction anesthesia. In the south and southwest and in smaller communities spinal anesthesia is felt to be almost synonymous with paraplegia. In smaller communities the patients often assume an obstinate attitude toward anesthesia and seem to feel that they should be allowed to select their own agent and technic. Any attempt at reasoning or conversion meets with resistance. This resistance is greatest when the anesthesiologist is just beginning to work in the small hospital. However, with the passage of time he will find patients becoming less obstinate and often friendly. The distrust of new things and new people will gradually disappear as the reputation of the competency of the anesthesiologist spreads in the community.

The evening before the contemplated surgery the anesthesiologist should make rounds before writing his orders for the following day. A personal interview with the patient will not only help to establish cooperation and confidence, but will often bring out some fact that the intern did not elicit. If the patient asks what type of anesthesia is to be administered, it is well to discuss it; otherwise I recommend that discussion be avoided.

In the south and southwest, it is best to avoid the word "spinal." I use the term "subarachnoid block" when the need arises and instruct the doctors and interns to do likewise. It sounds impressive and does not evoke the thought of paralysis. If abdominal surgery is contemplated I advise the patient that with subarachnoid block he will receive a light supplementary anesthesia to keep him asleep during the procedure. The universal antipathy toward conduction anesthesia for surgery seems to stem from being awake. Patients do not like to be awake and hear the tinkle and clatter of instruments while being worked upon. If, for some reason, it is necessary that the patient be awake, I inform him of this and explain that it is required by the surgeon. There is usually no opposition, especially when he is assured that I will be in constant attendance to take care of his needs and to make certain that the anesthesia is adequate.

If patients could be categorized, most anesthesiologists would probably classify them into two groups, "elective" and "emergency," each with three subgroups according to age: pediatric, adult, and geriatric. Some of these groups and subgroups present special features which permit the anesthesiologist to demonstrate to his colleagues the importance of his formal training.

In the elective group, the two commonest surgical procedures involve tonsillectomy in the pediatric group and gynecologic surgery in the adult group. The latter group does not pose any great problem to the anesthesiologist, and he can pick from a large variety of methods and agents. The tonsillectomy group can create many problems that the anesthesiologist can take care of to the benefit of the patient, the surgeon, and himself.

In many areas of the country it is common practice for the child to enter the hospital the morning of the day his operation is scheduled. This upsets all normal hospital procedure unless the child has pre-

viously had a thorough physical examination, history, complete blood count, urinalysis, and chest x-ray. This often is not enough, for if the parents have not been specifically instructed to the contrary, it is almost a certainty that they will have fed the patient a hearty breakfast in order to prepare him for his ordeal in the hospital.

In an attempt to solve this problem we have special admission forms with printed instructions for the parent. Here the parent is specifically instructed to make certain that all the items in the preliminary workup are completed at the time of the first hospital visit, including the visit to the business office. Then there are directions for the feeding of the child the evening before surgery, and in several differently worded statements the importance of not feeding the child after midnight is stressed.

A limit is placed for the time of admission. No child is admitted after 6:30 the morning of surgery. If the child arrives late, or there is evidence of previous feeding, the admission nurse is instructed to cancel the surgery and send the patient home until a boarding can be arranged on another day. After a few such cancellations, parents, surgeons, and other physicians will see to it that instructions are carried out, for eventually they will all realize that the rules were made for the safety of the patient.

In addition to administering the safest anesthetic to the child, I make every effort to contribute to the best working conditions for the surgeon and to obliterate postoperative psychic trauma both to the parent and the patient. Premedication consists of rectal Pentothal, plus demerol and scopolamine intramuscularly, each according to weight. When the patient is asleep, he is taken to surgery where induction of anesthesia is carried out with ethyl chloride or Vinethene and maintained with open drop ether until intubation can be effected. The surgery proceeds under endotracheal ether-air-oxygen, using a semi-open technic with the recently converted ether insufflator-aspirator. Constant suction in the oropharynx is used to aspirate all blood, thereby preventing it from seeping around the endotracheal catheter into the trachea. The eventual appreciation of the surgeon and the parent more than compensate for the extra time and effort that are required in administering this type of anesthesia to this group of patients.[4]

In the geriatric group of patients we have found the Evans blue dye determination of blood and plasma volumes to be one of the most valuable single preoperative evaluation tests. With the aid of the information derived from this test we have been able to correct deficiencies of blood volume and plasma volume accurately. Surgery is always deferred until they are brought into their respective normal limits. As a general observation of these patients so evaluated, it can be stated definitely that those who receive some form of spinal anesthesia have a more stable blood pressure and require fewer treatments with vasopressor drugs during the anesthesia. Also, as a group, these patients who have had their blood volume and plasma volume accurately measured and deficiencies treated are not prone to develop renal shut-down or shock during or after surgery. On this basis Evans blue dye tests have been made on all geriatric and poor risk patients, and the results have been gratifying. This service can be made available in any hospital, irrespective of the size.

ANESTHESIA FOR EMERGENCIES

In the field of emergency surgery, probably the commonest problem is that of fractures in patients who come to the hospital to have a closed reduction performed as an outpatient. No matter what the age, I feel that it is best to avoid general anesthesia if possible. For the upper extremity a brachial plexus block will supply very satisfactory anesthesia below the upper third of the arm.[5] For the lower extremity, a sciatic nerve block, with or without a femoral nerve block, will provide similar satisfaction.[6] The preference for conduction anesthesia is based on the fact that these patients too often have food in the stomach, and the reduction is usually carried out with fluoroscopic assistance. To date I have not learned of an x-ray room with the proper facilities for supplying general anesthesia in the dark safely.

The idea that children and very old people cannot undergo conduction anesthesia for orthopedic or other surgical procedures is unwarranted. In our hospital brachial plexus, sciatic nerve, and femoral nerve blocks, as well as many other forms of conduction anesthesia, have been administered satisfactorily to patients ranging from 3 days to 90 years of age. With proper premedication, conduction anesthesia in these cases has provided relief from pain during the orthopedic procedure, and has precluded the dangers of aspiration and laryngospasm.

The many other types of surgical emergencies do not present any special problem to the trained anesthesiologist in the small hospital. The one advantage of the large hospital over the small one is the ability to cope with hemorrhage. A source of blood is an urgent necessity. In its absence the anesthesiologist will find it difficult to improvise with plasma, dextran, or other blood volume expanders. For this reason the anesthesiologist must constantly lead the group with which he works to establish its own blood bank or else acquire the services of a community bank.

CONCLUSION

By following the tenets of good anesthesia practice, the anesthesiologist will find in a short time that he will be recognized as an important member of the hospital staff, and that he will experience a certain degree of satisfaction in meeting the challenges arising in the smaller institution. With properly kept records he can feel like a scientist in his own small laboratory. With all the factors of the department under his direct supervision failures due to inefficient assistants and uncontrolled equipment are at a minimum. The anesthesiologist will have found for himself, and will have proved to the staff members and their patients, that there is no dividing line between the small hospital and the large hospital as far as anesthesia service is concerned.[7]

5003 Ross Ave.

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Induced Hypotensive Anesthesia

Personal Experience with a Small Series of Cases

A. A. Golden, D.O.
Wilmington, Del.

PRELIMINARY REMARKS

Before beginning this paper I would like to make a few pertinent statements. It should be fully understood that I do not advocate this method for routine utilization by the physician-anesthetist. The hazard of morbidity and mortality far outweighs that of acceptable standard methods. *There exists an inherent capacity for catastrophe.* To illustrate: A recently published paper by Beecher and Todd[1] deals with a study of 599,548 patients anesthetized in ten university teaching hospitals. The death rate in this study from anesthesia was 1:1,560. In 21,000 cases in England and Ireland in which induced hypotension anesthesia was utilized in order to produce a relatively bloodless field, the mortality was 46, a ratio of 1:459.[2] The same authors studied 6,805 patients who underwent induced hypotension anesthesia in the United States; in this group there were 50 deaths, a mortality rate of 1:136.[3]

It is important, therefore, to keep these mortality rates in mind while reading this paper.

INTRODUCTION

As has been true so many times in the past, research in one line of endeavor leads to an accidental discovery of greater importance than the original object of the research study; it was so with the technic of induced hypotension by ganglionic blockade. While seeking a pharmacologic antagonist to decamethonium, Paton and Zaimis[4,5] discovered the hypotensive effect of the pentamethonium and hexamethonium compounds. The hypotensive effects of these drugs, resulting from their interference with ganglionic transmission, precluded their use as antagonists to decamethonium.

The importance of these compounds to the armamentarium of the anesthesiologist became apparent immediately. Clinical and laboratory experience produced a voluminous literature on the production of relatively bloodless fields for the facilitation of various surgical procedures.[6-12] With the accumulation of experience in this technic, a cautious feeling replaced the initial enthusiasm.

It is with this feeling of caution that I present my very small series of cases. Personal experience was gained from twenty-five cases, the patients varying in age from 17 to 72 years. There was no mortality, from either the surgery or the anesthesia. That may have been due more to good fortune than to skill, as when this series was being conducted, data came in from other anesthetic centers reporting unexpected deaths in supposedly good-risk patients. Intratracheal intubation was performed in all patients following the induction of anesthesia, since this is a requirement for the proper management of this technic.

PHYSIOLOGIC BASIS

It is felt that a brief explanation of the physiology and hemodynamics concerned with this "physiologic trespass"[13] technic will be of value. A hypotensive state during surgery is usually considered to be identical with shock—such as is seen following the loss of effective circulating volume and anesthetic depression. Accompanying this state is a compensatory vasoconstriction which, if not alleviated in a reasonable length of time, leads to tissue hypoxia and a resultant irreversible state. What, then, is the essential difference between low blood pressure induced deliberately by ganglionic blockade and that due to "shock" hypotension? Essentially, the difference is vasodilatation of the capillary bed. In shock compensatory vasoconstriction occurs, and in induced hypotension there is a block of the vasoconstrictor impulses with resultant vasodilatation.

Best and Taylor[14] list the following as factors in the maintenance of normal blood pressure:

1. Cardiac output
2. Peripheral resistance
3. Elasticity of the blood vessel wall
4. Effective circulatory volume
5. Viscosity of the blood.

It is the peripheral resistance or the vasomotor tone of the blood vessels that is affected with ganglionic blockade.

In hypotension due to shock there is a loss of effective circulating volume (as in hemorrhage) or fluid (by increased capillary permeability due to histamine release). The reaction of the body to this peripheral loss is one of vasoconstriction. Vasoconstriction causes an increase in peripheral resistance, and the work of the heart has to be temporarily increased to overcome this resistance. If the fluid loss is great enough, the heart cannot overcome this peripheral resistance and the blood pressure will fall. This lowered blood pressure, in turn, reduces venous return and cardiac output. As cardiac output is dependent on (1) the venous return to the heart, (2) the force of the heart contraction, and (3) the rate of the heart beat,[15] lowering or reduction of the venous return must be compensated for by the latter two factors. Hemorrhage and shock are characterized by an increased heart rate. Shock from a hemodynamic viewpoint, therefore, demonstrates a reduction in circulating blood volume followed by a compensatory vasoconstriction, while the hypotension has to be considered as a sort of exhaustion of the cardiovascular system in which heart function fails.

In hypotension due to blockade of the vasoconstrictor impulses there is peripheral vasodilatation, no loss of circulating volume, and little change in pulse rate. Capillary tension remains either normal or slightly above normal owing to loss of peripheral resistance.

In 1929, Cannon[16] deprived animals completely of their sympathetic nervous systems, and, though blood pressure fell, the dogs and cats lived in good health provided they were sheltered in the laboratory. Paradoxic as it may seem, the suggestion has been made that ganglionic blocking may be a shock preventive.[17]

TECHNIC

With the above in mind, it was decided to attempt induced hypotension for thyroidectomy. For a period of 3 years I had been using a nitrous oxide-barbiturate-meperidine-curare sequence followed by the insertion of a cuffed intratracheal tube. (The term "curare" refers to any of the curariform agents then in common usage.) This technic was nondepressant and nonhy-

poxic, and it was felt that the use of more potent agents such as cyclopropane or diethylether might intensify the hypotension produced by the ganglionic blockade. The decision was therefore made not to alter the technic in any way except for the addition of the required amounts of hexamethonium chloride.

Because of the vasodilatation produced by the ganglionic blockade and the resultant increase in the volume of the capillary bed,[18] it was felt that a slight reverse Trendelenburg position would further aid the production of a bloodless field by utilizing the effect of gravity. The height of the head seldom is more than 8 inches above the horizontal plane of the body.

An intravenous infusion of 5 per cent dextrose in water is started, preferably with an 18-gauge needle in a vein on the dorsum of the hand or wrist. The needle is securely taped. The blood pressure and pulse are then recorded. It should be ascertained that the stethoscope is firmly applied at the elbow and that the cuff is made secure.

Anesthesia is induced by an appropriate intravenous dose of 2.5 per cent thiopental sodium, given slowly, followed in most instances by a single injection of from 60 to 100 mg. of succinylcholine chloride. Following ventilation with 100 per cent oxygen for 30 to 40 seconds, tracheal intubation is readily performed in an atraumatic manner.[19] Several of the earlier patients were intubated with the aid of decamethonium bromide until succinylcholine chloride became available as an investigational drug. Following intubation, nitrous oxide-oxygen is administered in a 50:50 or 60:40 ratio, at a flow rate of not less than 8 liters per minute. With the return of spontaneous respiration, 25 to 50 mg. of meperidine are injected through the intravenous tubing.

The patient's pulse and blood pressure are then recorded, and he is positioned as mentioned above. Again the blood pressure is taken to note any alteration due to the posture change. It may be well to state here that while it is best for two anesthesiologists to manage the case in which the hypotensive technic is used, an adept and skillful individual can manage creditably.

The dose of hexamethonium is then given. Optimal results are obtained when the systolic blood pressure is between 70 and 80 mm. of mercury. The dosage of hexamethonium is determined by several factors:

1. The body weight. Invariably a heavier patient requires more of the drug.

2. The metabolic rate. When this is elevated, as in thyrotoxicosis, a higher dosage may be needed for successful results.

3. The lability of the circulation. This is assessed from the fall in blood pressure following induction of anesthesia with the ultrashort-acting barbiturate, and positioning. When these lead to a severe fall, as occurs in the hypertensive patient, a smaller dose of hexamethonium is given. When the blood pressure is but little affected by the induction, a larger dose of hexamethonium will be required. It is for this reason that injection of the barbiturate should be made slowly. It is a well-known clinical fact that rapid intravenous administration of an ultrashort-acting barbiturate may result in marked hypotension with accompanying apnea.

4. The age of the patient. The older patient, as a rule, requires a smaller dose than the younger individual.

The average dose of hexamethonium in the healthy adult male or female was found to be between 60 and 100 mg. The dosage utilized is roughly 1 mg. per kilogram of body weight. If the patient is hypertensive or more than 50 years of age, a smaller dose is given, but the ultimate dosage depends on his physiologic status. Within 5 minutes of the injection, dilatation of the pupils becomes evident, signifying parasympathetic block.

An early finding was that if the initial dose did not produce the expected result, further doses were of little value. The case of a 68-year-old male undergoing esophageal resection seems to bear out this contention. A total dosage of 140 mg. of hexamethonium was given at intervals in doses of 60, 40, and 60 mg.; 25 minutes elapsed before a drop in blood pressure occurred, and it was deemed at that time that the fall of the systolic blood pressure to 100 mm. of mercury was perhaps due more to decrease in the cardiac output from the pneophore intermittent positive pressure breathing technic than to the hypotensive agent per se.

With the desired blood pressure level obtained, the anesthesia is conducted in the usual manner, the administrator being on the alert from minute to minute to detect changes in the patient's status. When blood loss occurs it must be compensated for immediately since, with the compensatory vasoconstriction blocked, blood loss is poorly tolerated. The patient, as a rule, is awake and reacting on the operating table, albeit quieter and paler than with ordinary technics. Extubation is performed in the operating room after ventilating the patient with 100 per cent oxygen.

DISCUSSION

Certain important questions should come to the mind of every anesthesiologist contemplating the use of this method. What happens to vital organs such as the brain, heart, kidneys, and liver in the utilization of such a technic? Does this method have advantages over acceptable and proved methods? Does it further the patient's welfare?

When I first used this technic I did not have much laboratory evidence to help me, except for a few reports which had appeared in the British anesthetic literature.[20,21] It was felt if the patient were adequately oxygenated at all times that little or no serious changes would occur in vital organs. This has now been adequately proved by laboratory evidence.[22-28] It will be recalled by those familiar with the total spinal technic of the late Harry Koster[29] of New York, that he produced total spinal sympathetic blockade, placed his patient in a modified Trendelenburg position, and administered 100 per cent oxygen by mask. With this technic he performed thyroidectomies and mastectomies with no deleterious results to the patient. This fact is being mentioned at this point to illustrate that adequate oxygenation is of paramount importance.

As long as the systolic blood pressure does not fall below 60 mm. at heart level, the vital organs will be adequately supplied with oxygen. When the head

TABLE I—OPERATION STATISTICS (AVERAGES)

Age	Weight	Thiopental	Meperidine	Hexamethonium	Anesthesia Time
50.7 yrs.	64 Kg. (139 lbs.)	591 mg.	54.3 mg.	99.1 mg.	128.6 min.

of the patient is elevated, theoretically cerebral blood flow might be diminished. This seems to be an equivocal finding. However, oxygen uptake does decrease a little.

Sufficient clinical and experimental work has now been documented to demonstrate the effects of this technic on four vital organs—the brain, heart, kidney, and liver.[22-28,30] The cells of the cerebral cortex are the most sensitive to oxygen lack. This sensitivity can be demonstrated by changes on the electroencephalogram. Bromage[26] of England has demonstrated changes in the electrical pattern of the brain. Clinically I have not seen deleterious effects on cerebral function. The patients regained full consciousness in a normal manner and time; there were no changes in personality nor any degree of mental confusion. In 1953, Morris and his group[25] at Baylor University demonstrated a decrease in cerebral blood flow. Cerebral oxygen consumption, however, was only slightly decreased. Their conclusion was that there appears to be little danger of cerebral anoxia in hexamethonium-induced hypotension when the patient is in the supine position and the mean blood pressure is as low as 55 mm. of mercury. Dewar[31] recently showed that no significant change occurred in cerebral blood flow although the mean arterial blood pressure fell 30 per cent.

Most probably, the real danger of this technic is in the effect of the hypotension on the coronary circulation. Efficient coronary circulation depends on the mean aortic blood pressure;[32,33] during hypotension coronary blood pressure falls. Changes in cardiac output during the hypotensive episode produced by ganglionic blockade are variable, but observations have been made which indicate that it is unchanged or may be slightly increased.[34]

If the coronary circulation is healthy it is unlikely that coronary insufficiency will appear if hypotension is produced by ganglionic blockade; even though there is decreased coronary blood flow, the work of the heart and its metabolic needs are likewise reduced in proportion. If, on the other hand, the coronary circulation is impaired, or in some way does not partake in the general vasodilatation, it would seem that there would be a definite risk of coronary insufficiency developing despite the decreased cardiac work and flow resistance which follows the use of ganglionic blockade. That this is so is attested by the various reports from the literature.[2] Nevertheless, although the known case of suspected coronary disease may be ascertained by the history, it is a recognized fact that hidden coronary disease may exist, may be symptomless, and produce no change in the electrocardiogram.[35]

The kidneys have been the object of much research to ascertain the effect of induced hypotension on their function. It is a physiologic fact that glomeruler filtration is decreased at a systolic blood pressure of 70 mm. of mercury and ceases below 60 mm.[36] With ganglionic blockade it seems reasonable and logical to assume that in the presence of adequate oxygenation renal function should not be impaired. This has been adequately proved in normal man.[37] It must be remembered that the methonium compounds are excreted by way of the kidneys, and if renal impairment exists, this technic is interdicted. In the series of cases presented, anuria did not occur. In several cases, indwelling catheters were inserted so that the urine could be collected and measured. These observations lead to the conclusion that there is little or no disturbance of renal function by ganglionic blockade hypotension.

Bromage[26] studied the effects of induced hypotension on the liver. His series comprised seventeen patients undergoing upper abdominal operations. When the systolic blood pressure dropped to between 40 and 60 mm. of mercury, inspection showed cyanosis of this organ in fourteen of the seventeen patients, although tissues elsewhere were well oxygenated. It may well be that occult liver damage will result from this temporary hypoxia and that the systolic blood pressure should not be allowed to drop below a level of 60 mm. Several of the cases in my small series in whom Bromsulphalein and cephalin flocculation determinations were performed showed no significant difference in the values before and after hypotension. It may be concluded that, in my series, there occurred no apparent clinical disturbances of liver function.

Quite often the question of secondary hemorrhage is raised, especially after the blood pressure rises to normal or near normal. I have not seen any, although reports from other clinics indicate that a certain amount of secondary bleeding does occur. Cut vessels bleed, and the surgeon ties them as he would ordinarily. Capillary oozing usually does not occur, but, when present, is controlled by simple pressure. There must be scrupulous hemostasis on the part of the surgical team. The anesthesiologist should allow the blood pressure to rise gradually, without the use of vasopressors. Boyan and his group[38] use phenylephrine to return the blood pressure to normal. I prefer to allow the blood pressure to rise gradually without vasopressors, giving oxygen continuously by either BLB mask or tent.

SUMMARY

The impressions received from a small series of operations performed under hypotensive anesthesia have been presented. While my series is small, it should be explained that I assisted my associates in the management of many other cases. Provided its limitations and hazards are kept in mind, induced hypotension becomes a useful method in the anesthetic management of selected cases.

CONCLUSIONS

1. The use of a technic of such potential danger should be performed only by the expert anesthesiologist. The technic demands the undivided attention of the anesthesiologist, who must take repeated blood pressure estimations and carefully watch the pulse and appearance of the patient and at the same time keep in mind the possibility that the hypotension of shock may be masked by the hypotension of ganglionic blockade.

2. Patients should be adequately screened to eliminate any contraindications to this technic. Surgery must be such that the technic will be of aid to the surgeon and promote the welfare of the patient. To state the matter in another way, the anesthetic technic should not be more hazardous than the contemplated surgery.

3. Intratracheal intubation is mandatory to insure absolute control of oxygenation.

4. It should be kept in mind that there is an accumulation of evidence illustrating that drastic reduction in blood pressure with the methonium drugs carries a risk far out of proportion to that of acceptable anesthetic technics.

1009 Madison St.

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Caudal Anesthesia

ERNEST T. ROSS, D.O.
Okmulgee, Okla.

INTRODUCTION

Epidural anesthesia was discovered, apparently accidentally, by Corning in 1885. In 1895 Cathelin used epidural anesthesia in the sacral region. This is what is now known as caudal analgesia. The first book containing a thorough discussion of epidural anesthesia was written by Dogliotti in 1939.[1]

Epidural anesthesia, also called peridural and extradural anesthesia, is obtained by blocking the spinal nerves in the epidural space as they emerge from the dura before passing into the intervertebral foramina. Epidural anesthesia differs from spinal or subdural anesthesia in that the solution is deposited outside the dura instead of in the subarachnoid space.

There are three principal types of epidural anesthesia: (1) thoracic epidural block, (2) lumbar epidural block, and (3) caudal epidural block. It is with this last classification that this paper will deal.

ANATOMIC CONSIDERATIONS

Caudal anesthesia is accomplished by introducing a needle through the sacrococcygeal ligament and injecting anesthetic solutions into the caudal canal. The caudal or sacral canal is entered through the sacral hiatus which is a V- or U-shaped notch located between the sacral cornua formed by failure of fusion of the spine of the fifth sacral vertebra. The hiatus lies about 1½ to 2 inches above the tip of the coccyx, and the needle must enter the caudal canal through it. Puncture of the dural sac must be avoided, and the anesthetic solution must reach a determined level in the vertebral canal. The dural sac almost fills the vertebral canal, except for the epidural space which is bounded anteriorly by the periosteum of the vertebral bodies and posteriorly by the dura, and contains loose areolar tissue and blood vessels. The dural sac ends between the first and second sacral vertebrae, and the sacral prolongation of the epidural space is the sacral canal.

Topography is most important, and successful injection can be made easier if careful observance of landmarks is combined with palpation. The rhomboid of Michealis is very helpful in locating the site for the primary wheal. It can be visualized by imagining that straight lines are drawn bilaterally from the gluteal cleft to the dimples in the skin over the posterior-superior spines and from these dimples to the midline of the spine at the level of the fourth lumbar vertebra. The sacral cornua lie near the gluteal cleft dorsally and must be located prior to injection. After visualizing this rhomboid, palpation is used to find the cornua and the exact site for the primary wheal.

PHYSIOLOGIC PRINCIPLES

Anesthetic solutions disseminate quickly throughout the epidural space, and anesthesia is directly proportionate to the amount of anesthetic solution used;

for example, for low caudal anesthesia, to block the lumbosacral and coccygeal nerves, 30 cc. of solution are used. To produce midcaudal anesthesia, that is, block of the lower three thoracic nerves, about 60 cc. of solution are used. High caudal anesthesia, for block of the lower six thoracic nerves, requires about 90 cc. of solution.

The onset of anesthesia is slow and is first noticed around the sacral hiatus from which it spreads to the buttocks and sacrum.[1] Its occurrence within about 5 minutes indicates successful injection. Pain sensation is lost first; then the sense of touch and temperature, and after a total of 30 cc. have been injected, anesthesia will reach the umbilicus. Maximum anesthesia is reached in 20 minutes.

Anesthesia in this technic is more prolonged than spinal anesthesia and, after a single injection of 1.5 per cent procaine hydrochloride, usually lasts about 1½ hours. Metycaine, 1.5 per cent, also produces anesthesia which lasts for the same length of time.

INDICATIONS AND CONTRAINDICATIONS

Caudal anesthesia is most applicable to surgery of the perineum and extremities and is indicated in operations on the anus, rectum, perineum, and urinary bladder; in certain gynecologic operations such as those for cystocele and rectocele, perineorrhaphy, and vaginal hysterectomy; and in some obstetric procedures.

Contraindications to caudal anesthesia are:

1. Apprehensive patients who prefer being asleep and who may fear caudal anesthesia
2. Profound anemia and dehydration unless supplemented by oxygen and intravenous fluids
3. Easy short deliveries
4. Certain obstetric complications, such as placenta previa, abruptio placentae, pelvic disproportion, monstrosities and predetermined stillbirths, and sensitivity to drugs.

PREANESTHETIC CARE

In surgical procedures at the Okmulgee Osteopathic Hospital, the premedication routine, which, of course, is modified according to the patient, is as follows: One tablet of Carbrital is given at bedtime; 1½ grains of Nembutal at 7 a.m., and ¼ grain of morphine sulfate is given with 1/150 grain of atropine at 7:45 a.m. (45 minutes before surgery).

In obstetric cases, the following is the procedure: A preanesthetic enema is administered (at this hospital normal saline is preferred); Nembutal or Demerol is given 30 minutes before the anesthetic is administered. Engagement must be at station 0 or plus 1, contractions should last for 30 seconds and occur not more than 5 minutes apart, and dilatation should be from 3 to 4 cm.

CHOICE OF ANESTHETIC SOLUTION

The solutions used in caudal anesthesia are (1) procaine hydrochloride, 1 or 2 per cent; (2) Metycaine, 1.5 per cent; (3) Pontocaine, 0.15 per cent; and (4) Cyclaine, 1 per cent.

TECHNIC

In surgical procedures the patient is placed in the prone position with the hips slightly elevated and with the head against a pillow and the face turned away from the operator; the arms are not placed under the chest. (In obstetrics the Sims' position is used.) This position of the patient helps relax the erector spinae muscles and makes the injection easier. The usual sterile precautions and technic are observed. After the site for injection has been carefully selected, a skin wheal is made with a solution of 1 per cent procaine. Common mistakes in locating the site for the preliminary wheal are to make the injection either to one side of the median line or too far cephalad in the median line. The wheal can more easily be made if the bevel of the needle is placed against the skin and held so as to force it into the superficial layers of the skin. This is the reverse of the usual method.

Caudal anesthesia may be produced by administration of either a single dose or as a continuous or fractional dose. The caudal canal will tolerate a total of 25 cc. at one time so that if injection of a greater amount is needed, there must be a lapse of 30 or 40 minutes to allow for dissemination and absorption.

For the preliminary injection into the caudal canal a medium-caliber needle about 50 mm. long is guided through the prepared wheal into the canal by way of the sacral hiatus. About 5 cc. of anesthetic solution are injected. A flexible or malleable 19 gauge spinal needle is grasped by the thumb and middle finger with the index finger resting on the stylet end. The wheal needle is removed, and the second needle is inserted into the sacral canal through the same puncture. It should be kept at an angle of 45 degrees with the skin and rotated through 180 degrees as it is injected. If bone is reached, the needle is withdrawn slightly until the point clears the bone, and the angle is changed so the needle is parallel to the long axis of the canal. It is advanced no higher than the second sacral vertebra (this distance is measured beforehand). Gentle massage may help to advance the needle more easily.

If, on gentle aspiration, blood appears, the needle is withdrawn slightly and aspiration again performed to ascertain that injection is not intrathecal or intravenous, either of which will produce serious consequences. Strict adherence to these precautions will give reasonable assurance that injection is being made into the correct space. If spinal fluid appears, the anesthesia must be discontinued.[1,2]

If no blood or spinal fluid appears when the stylet is removed, a 10 cc. syringe, which should contain slightly less than 10 cc. of anesthetic fluid, is attached. Aspiration should be repeated. Next 5 cc. of anesthetic fluid are injected and aspiration is repeated again. If no blood or spinal fluid appears, the test dose of 8 cc. is completed by slow injection. After waiting to make sure no untoward effects appear (approximately 10 minutes), the anesthetist continues the injection until the desired anesthesia is produced.

After a total of 25 cc. of anesthetic solution has been injected, the caudal needle is left in place and, after 30 minutes, another 5 or 10 cc. are injected. Satisfactory anesthesia should be attained if the proper technic has been strictly adhered to.

If the caudal needle is behind the sacrum, tumefaction will result, the needle having been injected at too great an angle with the skin. If, on gentle aspiration, spinal fluid is obtained, the needle is in the subarachnoid space. If blood is obtained, the needle is in a vein. No harmful effects occur if the needle is inserted in front of the sacrum; however, anesthesia is not obtained.

Signs of Anesthesia.—Signs that the injection is being made into the caudal canal are: Tingling and hyperesthesia referred to the thighs and legs occur. There is a sensation of pressure over the sacrum. Anesthesia over the coccyx develops quickly. Respirations increase and the patient sighs because of the pressure of the solution. When this occurs, injection should be stopped for 30 seconds, after which respiration should be re-evaluated and injection resumed.

ADVANTAGES AND DISADVANTAGES

Advantages of caudal anesthesia are:

1. The risk in comparison with that of general anesthesia is negligible.
2. Postoperative sequelae are absent if the technic is perfect.
3. Postspinal headache, hypotension, and nausea are infrequent.

Criticism of caudal anesthesia because of the complexity of the technic is not wholly justified because the technic can be mastered by most individuals in a short time.

The disadvantages of caudal anesthesia mainly stem from the fact that many patients are unfit for caudal anesthesia because of anatomic defects and anomalies. Detailed studies[1] have shown that:

1. In 40 per cent of cases, the dural sac extends below the middle of the second sacral vertebra.
2. In 42 per cent of cases, the distance from the dural sac to the hiatus is less than 47 mm.
3. In 47 per cent of cases, the apex of the hiatus is at a higher level than the lower third of the fourth sacral vertebra.
4. Accessory apertures are present in 25 per cent of cases.
5. Agenesis of the dorsal wall occurs in 2 per cent.
6. The anteroposterior diameter of the canal at the apex of the hiatus is 2 mm. or less in 5 per cent.
7. In 1 per cent of female pelves there is no hiatus because of fusion.
8. Blocks of the lumen of the sacral canal are present in 1 to 2 per cent of cases.

However, only about 10 per cent of these are of significance from the standpoint of caudal anesthesia.

Blocking the sacral nerve roots abolishes the pain resulting from distention of the birth canal. This must be kept in mind in order to prevent inadvertent overdistention of the bladder in obstetrical patients. Care must also be exercised in transurethral manipulations for prostatectomy and in fulguration of vesical tumors.

SUMMARY AND CONCLUSIONS

Caudal anesthesia, its anatomic and physiologic principles, its indications and contraindications, and its advantages and disadvantages have been discussed. Solutions used have been mentioned, and preanesthetic care and the anesthetic technic have been described.

Caudal anesthesia should be used as a prescription anesthetic for certain cases and certain indications; it should not be used routinely in obstetrics. The technic itself has from 10 to 20 per cent of failures and is indicated only when the need for protracted periods of anesthesia is anticipated. However, it should not be used for procedures of too long duration as the incidence of failure increases sharply after 8 hours.

420 Commerce Bldg.

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Psychiatric Aspects of Anesthesia

RALPH IRVING McRAE, B.A., B.Sc., D.O., F.A.C.N.
Dallas, Tex.

In few fields of medical progress has there been the advance of technical skill, splendid flexibility, and yet accurate specificity of action as is seen in the field of modern anesthesiology. Within a slightly longer time than that in which the osteopathic profession was conceived, given birth, and grew to its present adolescence, this technical skill in relieving the anguish of pain and its attendant reactions has come to its maturity. Like other young adults, its future goal is the achievement of wisdom. The transition from the skilled technician to the subtle understanding artist is to be achieved by those whose knowledge and wisdom combine to form a synthesis of intuitive perspective, in which the human side of the patient is perceived as an over-all background for evaluating the physiologic status and the psychologic reaction to the anesthetic.

It is the peculiar function of the anesthetist to separate the psyche from the soma. Therefore, it is not asking too much that he should become acquainted with the psyche as well as the soma in dealing with his patient.

It is a striking paradox that the anesthetist, who in a very real sense carries the patient's life in a delicate balance, suspended between coma and death, is given the least opportunity to study his patient, is the least recognized for his true responsibility, and too often cares too little to get involved with the personality of his patient.

Too often, as the technician administers his liquids, gases, and injections and modifies his set of shiny gadgets to control changes, the patient is only a set of reactions which follows in certain stages—a pattern of bodily changes. Too many anesthetists act as if all people were born free and equal, a fallacy of considerable magnitude in law, and one which should never be held in the field of medicine.

The crucial question is how important is it for the anesthetist to know, and know how to manage, the psyche as well as the soma of his subject. Put another way, how important is it to know the condition of the psyche at the time it is divorced from the soma, and what can be done to insure its tranquillity?

Few realize the tremendous impact that the few ounces of anesthetic agent have on the total personality of any patient when it divorces these, its basic components, while the soma is being subjected to scalpel, traumatic handling, the hemostat, and the needle. Too glibly the textbook speaks of the anesthetic acting from above downward in the brain and from below upward in the spinal medulla. This is an invasion of the integrity of a relationship which has to do with the depths of instincts and aspirations of creative intellect.

This is not a maudlin philosophic or metaphysical dissertation. This is a crucial practical problem which must be recognized and comprehended. It is a fundamental tenet of psychiatry that traumatic events are

enormously qualified in their impact upon the personality by the condition of the psyche at the time of the traumatic experience, that is, if the psyche is in a state of calm, serene understanding preparedness, the impact of surgery and the transition from and back to consciousness will have few complications or sequelae, but if the psyche is tense, frightened, and intensely confused emotionally over the organ to be operated upon, the induction, recovery, and sequelae can be expected to be stormy at the personality level, and may be disastrous to basic integrity or sanity.

It is well to keep in mind that both consciousness and unconsciousness are the domain of the psyche. To disassociate this psychic component from its somatic relationships when in a state of acute distress is highly traumatic and greatly magnifies the impact of any misadventure of surgery or convalescence, or it may disorganize personality function for years to come, even if there is no misadventure clinically at the time of hospitalization. It is another striking paradox that the public fears surgery and glorifies the surgeon for his skill, yet does not recognize that it is the anesthetic that produces the fear and trauma to personality and that the anesthetist is the one whose skill protects the patient's life, while the surgeon carries on skillful, but purely mechanical, procedures upon somatic tissues.

It is an even greater paradox that the anesthetist is often just as unaware of his responsibility in regard to the pathologic effects of the procedures upon the total personality of his patients, because he does not inquire into the condition of his patient's emotions before, or does not follow up after, anesthesia.

The problem is complicated by the fact that the surgeon, because he is an expert in handling and excising physical tissues under the anesthetic isolation of the patient's personality, also has a blind spot relative to this problem. The patient is elected to go to surgery for a variety of reasons, few of which take into consideration the psychologic aspects of the patient or of his surgical problem. For this reason the anesthetist has often to perform his task of preparing a patient for surgery when neither he nor the surgeon knows the state of the patient's mind and emotions at the time of basal anesthesia or during induction proceedings. Disturbed behavior of the patient is as often blamed upon the type of anesthetic as upon his emotional condition.

Some elementary case problems may clarify thinking on this situation. In the renaissance period of spinal anesthesia it was a smart stunt, and this was not many years ago, to permit patients to watch their own abdominal or pelvic surgery in a mirror. I know personally of patients with this history who had to have a series of electroconvulsive treatments, which were preceded by months of psychic anguish and of vivid imagery in sleep and wakefulness. Psychiatry has for years been impressed with the case histories beginning with, "Ever since my surgery (or a specific surgery in a series of surgeries), I've had trouble." "That is when it all began, just after my hysterectomy." Another group of patients puts it this way: "I didn't want the surgery; they frightened me and I agreed. I was very tense when I woke up that morning, and the shot they gave me didn't seem to help much. When I came out from under the anesthetic I felt I was changed; I didn't feel the same any more. That was 5 years ago, and I've been getting worse ever since."

These are not isolated cases. This is a common problem in men and women of all ages seen in psychiatric practice. Constant rechecks reveal no local problem in the surgical field, yet the patient is seriously altered in total well-being. There are too many very successful similar surgeries to blame the problem on the surgery itself. It is not enough to say the patients were neurotic or unstable, because the date of the change is after the recovery from the anesthesia. Before that they carried their responsibilities well. It is revealed in their histories that the anesthetic disorganized personality when the psyche was in great turmoil and the surgery was longer in duration than average.

The skill and wisdom of anesthesiology will be marked by the recognition that the state of the psyche at the time of basal and surgical anesthesia is a vital key to successful smooth induction and freedom from postsurgical psychopathology.

Because of the anesthetist's peculiar isolation on the surgical team, the patient has little access to him and, as a result, the anesthetist has little realization of the postanesthetic personality changes or neurologic difficulties that do occur. There are still large numbers of people who go home, tip the nurses, and do all the proper things, and never speak of the anguish, the long-delayed recovery of strength, or other common sequelae of anesthesia. These sequelae are usually not recognized even by the patient as having any specific relationship to the anesthesia, but careful study, and the evaluation of details in the factual history of the hospital stay, clearly point to the problem's being centered in this area.

ANESTHESIA IN CHILDHOOD

If the impact of anesthesia in adults can be so disturbing, it is obvious that the effect upon children can be even more devastating. The normal infant and early childhood personality is inherently unstable in its basic integration. The putting together of body, mind, emotions, and the environment into meaningful patterns is a long and complicated process. It only begins to become somewhat coherent around the fifth to seventh year in normal children.

The separation of psyche from the soma in the preschool years is a hazardous project at best. The traumatic effect is greatly increased when there is confusion, struggle, fear, or panic in the youngster's mind at the time of anesthesia. The earlier in life the event occurs, the greater is the psychologic impact of the total surgical procedure.

Acute psychologic symptoms are seen in as many as 50 per cent of children who are operated upon in the first 18 months of life. This drops from 50 per cent to 8 per cent by the seventh or eighth year.[1] The fact that most childhood surgery is directed at two critical somatic areas of personality, the throat (tonsillectomy and adenoidectomy) and the genitalia (circumcision) is an important factor in the genesis of psychopathology. This, however, only places a greater responsibility upon the anesthetist to protect young patients as much as possible from any and all disturbances of the psyche in his over-all management of them.

PSYCHODYNAMICS OF TONSILLECTOMY AND ADENOIDECTOMY, AND CIRCUMCISION

What is meant by "two critical somatic areas of personality, the throat and the genitalia"? The first act at birth which is so gladly hailed is the wail of the newborn. The next major activity of the infant is that of maintaining life by sucking and swallowing. This is a complicated activity which is totally satisfying and therefore meaningful to the early dawning sense of the ego, or self. Around the mouth and throat the personality has its origin.

In early infancy the use of the throat for crying is the only defense, the only language, the only command the child has over its environment. Feeding continues to canalize pleasant, satisfying meaning around the activities of the mouth and throat. They therefore become a two-way source of satisfaction and power by means of swallowing and crying, and thus are the primary centers of personality organization. As smiling, talking, whistling, and kissing are learned, this area of somatic structure becomes the center of personality function and continues as such throughout life.

In childhood the impact of the anesthetic cone and the procedure of tonsillectomy and adenoidectomy has been found to be a serious threat to ego strength and personality stability. Circumcision, when done quite early in infancy, may not be so traumatic, inasmuch as sensibility is still low in the genital area, and personality has not included this area in its perimeter in the first few days of life. After some toilet training and the dawn of consciousness of the infant or child of the special sensations in the genital area, the impact of circumcision may become a very real source of psychic conflict. Here anesthesia, basal, surgical, and postsurgical, should be adequate and effective. Too often the procedure is done as minor surgery, with several people holding the child down, often with a parent helping. In spite of all that Freud[2] has demonstrated and confirmation of his findings by workers in all parts of the world, it is still difficult for physicians and laymen to understand the pivotal significance of the sex organs, especially of the male, in infancy and childhood.

Certain obvious facts may help clarify this problem. First, children become acutely aware of their bodies early in life, on a basis of sensory stimuli. The erogenous zones develop quite early and constitute a common source of pleasure. The most sensitive of these are the lips and the external genitalia. The genitalia obviously develop a special type of pleasant sensation as indicated by the advent of masturbation movements early in development.

Second, throughout infancy and childhood in our culture, the sexual organs are the only parts of the body that are always kept covered, except at the time of bathing or diaper change. Third, in young children there are no physical differences between the sexes other than the genitalia. Here is a basic dichotomy of the "haves" and the "have nots" that is a source of enormous pivotal difficulties which are to be projected throughout life. Fourth, there is only one early, central, and strongly emphasized area of "training" in early childhood, the area of toilet training. This is the first time the child has the opportunity to dominate the inter-relationship of parent and child, and he does so by giving or withholding his own bodily products. The anal area comes under control almost automatically. The urinary area, especially in boys, is notoriously resistant to control.

Another vital factor in this drama of sex in the young child is the all-important overpowering significance of sex differences between its image of mother and father. The soft, warm support and primary comfort of mother's femininity is the soil in which the personality took its roots. The rough, clumsy, intermittent impact of the father-figure comes first as a threat and an irritability to be adjusted to and coped with. Father is strict, punitive, and arbitrary. He also excludes the baby from the mother's side at night when the infant is the most hungry or the most frightened or the most alone and helpless. Young children are bundles of perception. They have little else to do but perceive and put things together as best they know how. Somewhere between 18 months and 4 years of age, the relationship of external sex organs to motherness and fatherness comes to a focus. At this point the concern over having or not having these appendages becomes a source of deep concern. Questions of possibly losing them or of somehow getting them (in the case of girls) becomes a source of emotional difficulty.

Concurrently the clothing begins to differentiate the sex of children, and the association of these organs to the entire drama of life becomes more obvious and a subject of more confusion and anxiety. If in the middle of this growing confusion over "have" and "have not" the child is subjected to belated circumcision, the concept of a castration complex becomes quite real and tangible. Such conflicts lay the foundation for many disturbances in the emotional life of the patient for years to come.

SENSITIVE PEOPLE

Next to young children, the sensitive people—the allergic, the hyper-reactive, the neurotic, and the unstable young adults—present the greatest hazards in terms of psychic disturbances following anesthesia.

A little knowledge can be a dangerous thing, and as anesthesiology goes through its own developmental stages, certain evidence is seen of this problem of a "little knowledge." From the attitude of not being responsible for differences in personality, many anesthetists have gone to another extreme of heavily sedating all patients to avoid any problems that may arise in getting the patient into a satisfactory level of anesthesia. This is oversimplification and may be a serious threat to some. Another tendency commonly seen is that, if the anesthetist decides in his own mind that he is dealing with a hysterical or otherwise neurotic personality, he will avoid all problems by administering heavy basal medication. Unfortunately, those who look frail and give the impression of being emotionally unstable are also easily intoxicated by the barbiturates and synthetic narcotics now in vogue. Their central nervous systems are not only sensitive, they are also prone to allergies and react with major distress to many drugs and some anesthetics, such as cyclopropane, with almost disastrous results. Many of these people are fully aware of their own sensitization and have avoided drugs all their lives with very real and well-founded fear. To them, heavy hypnotics, taken the night before at the authoritative insistence of the night nurse, and heavy basal medication are a threat. A state of consciousness is produced that is very close to a major schizoid state and, at the same time, it disorganizes the central nervous and autonomic reserves. When to this is added the major anesthesia for which these patients may have poor tolerance, the effects, in terms of shock, slow arduous recovery, and central nervous system dysfunction, may easily outweigh all benefits derived from elective dilatation and curettage or other more extensive procedures. It behooves the anesthetist to inquire into and study carefully the history of the patient's reaction to medication. It is important too to differentiate between the neurotic and those who have poor constitutional capacity. They may or may not be coupled together. When they are concurrent, it is advisable for the anesthetist to handle the patient on the basis of his constitutional inadequacy rather than to defend himself against the patient's neurosis by excessive sedation and poorly selected anesthetics.

CONCLUSION

In conclusion, it should be emphasized that the anesthetist has a real professional responsibility to do

everything possible to insure a smooth, uneventful, and predictable program of preparation and transportation of the patient to and from surgery. It is fundamental to human nature that if things are following through on schedule confidence, peace of mind, and a minimum of emotional disturbance are engendered. The presurgical patient is in a normal state of psychic tension in any case. Delays, confusion, arbitrary management, careless handling, unguarded conversation, bad language, and the failure to keep the patient posted as to what is expected of him, all conspire to increase this tension to a critical point.

It is important to recognize that the ego strength, degree of integration, and quality of personality stability are essentially good in a large number of patients who have a genuine surgical problem. However, there is an increasing number of sensitive, allergic, and inadequate patients who require special management on the part of the entire staff. They particularly require special handling by the anesthetist. It is also the responsibility of anesthetists to increasingly modify and influence the organization of their hospital to the end that the patient arrives in the operating room in a tranquil, passive, and cooperative state of mind. All disturbance in the hospital should be eliminated so that the patient feels he is in the hands of a competent, efficient, cooperative team of specialists who are endeavoring to insure his personal welfare, comfort, and safety.

Elective surgery in children should be performed in the first few days of life or postponed until they reach school age. In handling children, it is necessary to take the time to help them feel they understand what is to be done and to have a good reason in their minds for doing it. Children should be protected from fear, pain, and confusion, but particularly from fear and confusion. Most people can accept a great deal of pain if they can understand it, but all people are greatly disturbed by fear and confusion. Patients should be dealt with on an individual, personal basis; and the anesthetist should listen intently to what they say and should pay attention to their hands and eyes, as these often reveal what patients are feeling and fearing.

This does not mean that it is advisable to spend a great deal of time with the patient. It is important only to use intuition and then to check the conclusions scientifically by careful questioning and observation to see if they are right or wrong or if a significant fact about this patient and his peculiarities has been missed. As his technical skill is perfected, the anesthetist should strive to develop a unique intuitive capacity for judging the status of his patient, remembering always that the patient is a singular, all-important personality whose psychologic state will remain the same or be very much worse, for a long time, for having been under the anesthetist's care. With the splendid achievement of his technical skill the anesthetist should be secure enough to face patients as they are, not as he hopes they are, or is afraid they are, or as if he does not care what they are. The rising dignity of the specialty of anesthesiology behooves anesthetists to achieve the true maturity of wisdom.

6621 Snider Plaza

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Blood Volume in the Surgical Patient

J. F. DePETRIS, D.O.
Dallas, Tex.

Today there is general agreement that it is mandatory to maintain an adequate total blood volume and its relative constituents in order to prevent and eradicate the medical catastrophe known as shock.[1-5] Reduction of blood volume due to hemorrhage or skeletal trauma is the most common cause of death during the first 48 hours following injury.

Recent evidence using blood volume studies shows that reduction in blood volume in shock patients is fully accounted for without assuming that there is general leakage of plasma from the circulation through increased capillary permeability.[6-8] In the Evans blue dye (T-1824) method of blood volume determination, shock does not cause any profound change in the dye curve, thus demonstrating that increased capillary leakage is not an essential feature of shock. It is also borne out by the fact that the rate of dye loss from the plasma is essentially the same whether or not the patient is in shock. Marked loss in plasma volume may occur in burns and peritonitis where direct damage to a local capillary bed occurs.

Fluid is actually absorbed by the blood stream in the uninjured regions and tends to compensate for the loss of blood at the site of injury. This is entirely in accord with the common clinical finding that patients in shock show a tendency toward hemodilution rather than hemoconcentration.[5]

In cases of hemorrhage and skeletal trauma the train of events leading to shock is initiated by a large reduction in blood volume.[4,6] In both dog and man an acute reduction of 30 to 40 per cent in the circulating blood volume is followed within 1 to 2 hours by the appearance of the characteristic symptoms of shock.[2,4,5,8] Skeletal trauma is associated with the formation of a large hematoma at the site of the injury; in surgery the loss of whole blood is from hemorrhage; and with burns or in peritonitis loss is due to plasma filtration from damaged capillary beds.

However, one must not lose sight of the fact that shock in the surgical patient does not always mean diminished blood volume. This may be the case in head injuries, in cardiac tamponade from pericardial hemorrhage, and in myocardial infarctions or failure. In such shock, especially myocardial failure, blood volume determination is an aid in differential diagnosis and will demonstrate whether blood volume needs to be in-

creased. The surgically significant feature of reduced blood volume is that there is an increased susceptibility to shock which can be corrected by transfusion replacement of the blood volume deficit.

A state of chronic shock has been described in which the usual 1:3 ratio of plasma volume to interstitial fluid volume is altered owing to the decrease in plasma and increase in interstitial fluid seen in the chronically ill patient.[2] Chronic shock is defined as a syndrome characterized by weight loss, diminished blood volume, and increased interstitial fluid volume, which results in an increased susceptibility to shock from anesthesia or minor operative trauma.

The presence of reduced blood volume in a patient with weight loss is confirmed by the demonstration of restoration of blood volume to a higher level during convalescence. Recognition of the factor of tissue protein depletion offers a mechanism whereby the interstitial fluid volume could expand as the result of reduced tissue tension associated with loss of tissue protein, such as collagen. It has been shown that weight loss is correlated with the reduced total mass of circulating plasma protein even though its concentration is not significantly altered.[2]

There is a definite ceiling on the rate of synthesis of new hemoglobin and red cell regeneration which may be inadequate for the surgical exigency in a chronically ill patient. In many cases operation cannot be wisely postponed until the patient has synthesized adequate quantities of both hemoglobin and tissue protein. In the absence of anemia tissue protein regeneration is accomplished rapidly. In the chronically ill anemia may be severe, but reduction in plasma volume alters the hematocrit concentration, and it fails to give a true picture of the existing deficiencies in hemoglobin and red blood cells.[9,10]

It is of interest here to cite statistical results obtained by Clark and his associates[2] in the estimation with T-1824 of the standard circulating blood volume of chronically ill patients with weight loss. Eighty-nine patients were studied. Average blood volumes for the various groups, expressed as per cent of standard for usual weight, were as follows: malignancy, 72 per cent; hepatic disease, 67 per cent; chronic infection, 76 per cent; malnutrition, 76 per cent; and peptic ulceration, 79 per cent. The deficit in total blood volume is approximately one-fourth the value accepted as standard for the patient in health. The average total hemoglobin ranged from 50 to 60 per cent of the standard, and the average total red blood cell mass ranged from 56 to 70 per cent of the normal.

It has long been recognized that blood volume depletion occurs in patients with neoplastic disease.[2,11,12] Ewing[11] quotes Louis who in 1846 stated that the total quantity of blood was usually very reduced in patients with malignant disease. The major factor for this reduced blood volume has been shown to be a deficiency in the total circulating red cell mass and in circulating hemoglobin. Plasma changes are slight.[2,5] Kracke[13] stated that malignant processes within the body either elaborate a product capable of bone marrow inhibition or utilize substances necessary for red cell production.

Hemoglobin concentrations in grams per 100 cc. of blood may be normal and indicate little or no need for blood replacement, but if the total circulating hemoglobin is calculated, a patient with 13.4 grams of hemoglobin per 100 cc. may have an actual total hemoglobin deficit of 330 grams and require 2,200 cc. of whole blood to replace this deficit.

Similar mistakes may be made in erythrocyte and plasma volume estimation if only the red blood cell count or hematocrit determination is used, as these are only concentration values and not total circulating mass volumes.

Studies by Stanton and his coworkers[14] on operative and postoperative blood loss, using the Evans blue dye method, demonstrated that the previously used gravimetric and colorimetric methods are inadequate and that the loss is greater than previously found. Also, further blood loss may occur in the postoperative period and exceed the actual operative loss by 100 to 200 per cent. Blood loss is often noted, as in the sanguinous drainage following urologic operations or in the guaiac-positive stools after gastrointestinal resection, but this does not account for the commonly found deficit of red cell mass in many postoperative patients. The observed postoperative decreases in red cell volume cannot be adequately explained by either excessive compensatory plasma dilution or changes in the mean corpuscular volume of the red cells. Increased or abnormally rapid destruction of transfused red cells does not usually occur, nor could excessive blood destruction or decreased blood formation be blamed, as these were ruled out by appropriate studies including icteric studies, bilirubin studies, and reticulocyte counts.

Patients with peritonitis have little change in the red cell mass, but reduction in plasma volume of 20 to 30 per cent may occur.[15]

Replacement of severe uncompensated depletions in red cell volume as a result of surgery takes place only after several weeks.[14,16,17] In contrast, in the adequately hydrated patient, restoration of the plasma volume and total circulating protein to or above average control values occurs in 1 to 3 days.[16] Therefore, since plasma and cells are lost in the same proportion as they are present in the blood at operation, the hematocrit determination immediately following surgery may be essentially unchanged from that preoperatively. However, once the plasma volume is restored the actual red cell loss becomes fully apparent in the decrease in the hematocrit reading on the third day after operation. Here again T-1824 blood volume study is more helpful.

Because plasma is quickly replaced and the body cannot without aid rapidly repair severe red cell volume depletions, the major role of blood replacement therapy in the postoperative period is to replenish the depleted red cell mass.

A critical appraisal of blood volume estimation procedures in clinical use leads to consideration of a method which is not too technical for the average laboratory personnel, one which is not overly time consuming, and, most important, a method with a percentage of error within limits small enough to permit proper evaluation and control of chronically ill patients. It is generally conceded by most investigators that a means of determination of the blood volume which meets these criteria completely is not as yet forthcoming.

The most widely used technic for estimating plasma volume is that using Evans blue dye.[9] This dye is rapidly bound to the serum albumin, and therefore very little will leave the circulation. An accurately measured volume of the dye is injected intravenously, and at the same time a dye-free blood specimen is drawn. Ten minutes later a second specimen of blood is removed from the opposite arm. This time allows adequate mixing of the dye and only insignificant

loss from the blood stream. The plasma volume is determined by measuring the amount of dye dilution with a photoelectric colorimeter. The blood taken before the introduction of the dye is used for hematocrit determination, which must be corrected using the factor 0.96 for plasma trapped among the packed cells.

First the total plasma volume is estimated, and then the total blood volume is calculated from the plasma volume and the corrected hematocrit. The total circulating red cell volume equals the total blood volume minus the plasma volume. The total circulating hemoglobin may be estimated from the total blood volume and hemoglobin concentration. The total plasma protein is estimated from the total plasma volume and protein concentration of the plasma. This procedure may be done in any small hospital laboratory in about 30 to 40 minutes with a minimum of equipment and expense to the patient.

The problem of determining a depleted patient's normal weight as the basis for replacement is not yet completely settled. For the individual of average body configuration, this presents little difficulty. An average blood volume of 85 cc. and a plasma volume of 45 cc. per kilogram of body weight is fairly well established. In abnormally obese patients and unusually thin individuals, however, an accurate basic weight cannot easily be determined. Normal values are then calculated on the basis of average weight of individuals of the same age, sex, and height as derived from prepared charts.

Various methods have been utilized for determination of plasma and total blood volumes. Most have depended upon dilution of the blood stream by some introduced material, such as foreign proteins, vital dyes, gum acacia and hemoglobin, carbon dioxide, radioactive iron, chromium- or phosphorus-labeled red cells, or iodinated (I^{131}) human serum albumin. Since it is impossible to measure directly the plasma and blood volume in a series of normal individuals, there are no absolute values available for determining the accuracy of any given indirect method.

Brady and his coworkers[18] simultaneously compared the plasma and total blood volume in the same patient by using radioactive phosphorus-tagged red cells, iodinated human serum albumin, and Evans blue dye. The first test gave the lowest values, the second the middle values, and the third the highest. By using the above data it was demonstrated that the hematocrit determination for the total body is lower than the hematocrit determination for the peripheral vessels. Evans blue dye and iodinated human serum albumin values for direct plasma determination on the same patient were reproducible and equally accurate, varying only 0.31 ml. per kilogram of body weight.

Root and his associates,[19] using the factor 0.96 to correct their hematocrit determinations for plasma trapped among packed cells, found the ratio of the total body hematocrit reading to be lower than the peripheral vessel hematocrit reading. In their opinion their results do not disprove that the cell-to-plasma ratio is less in the capillaries than in the large vessels, but show that the amount of blood in the small vessels was not large enough for the unequal red cell distribution in these vessels to affect greatly the estimation of the blood volume as calculated by the Evans blue dye and hematocrit reading under these conditions.

Meneely and his group,[20] using Fe^{59}-tagged red cells and Evans blue dye and using the hematocrit correction factor of 0.96, and Berson and Yalow,[21] using iodinated serum albumin and P^{32}-tagged red cells and using a hematocrit correction factor of 0.98, came to similar conclusions.

Theoretically, since both are direct measurements and do not introduce the error of the peripheral vessel hematocrit determination, it is better to determine the total blood volume by adding the red cell mass determined by radioisotope-tagged red cells and the plasma volume obtained with iodinated human serum albumin. However, this is a literal impossibility in the small osteopathic hospital whereas the more practical Evans blue dye method is not.

Using the Evans blue dye-hematocrit method to redetermine the plasma volume in the same individual, the error is less than 5 per cent.[3,22] I believe that, compared with the routine laboratory tests used today, determination of blood volume with T-1824 and calculation of deficits as recommended are reasonably accurate clinically and constitute the most reliable test available for the small hospital.

In evaluating the blood volume in the surgical patient, not only is the total volume important but the blood constituents should be properly assessed. The following procedures should be done: hemoglobin concentration, hematocrit determination, erythrocyte count, and protein concentration. Blood volume determination with Evans blue dye will give the following information: total plasma volume, total blood volume, red cell volume, total plasma protein, and total circulating hemoglobin.

It may be argued that even these simple laboratory tests are not needed for intelligent care of the patient, because whole blood and proteins may be given as his clinical appearance seems to indicate. Emphatic exception must be taken to this attitude with the reminder that it is not uncommon to find a serious deficit in persons who have not lost weight and who may appear to be reasonably good operative risks. Experience has taught us that while clinical impressions are often accurate, they may be disastrously misleading and should not be relied upon solely.

Fear has been expressed regarding blood replacement in the presence of cardiac disease. Quantitative replacement is, I believe, more important in such cases than in those patients without heart disease and may make the difference between death and a successful outcome. Solutions containing sodium, however, should be given with caution.

It must be remembered that a small deficit in a small individual is just as serious as a large deficit in a large individual.

The quantitative replacement of the blood volume deficit preoperatively and blood loss during surgery will almost eliminate operative and postoperative shock,[1-3,11,16,23] widen the scope of surgery, and lower mortality and morbidity. Postoperative convalescence has been rapid in patients thus treated as compared with the slow recovery of many patients not receiving replacement. Lyon and his associates[16] have shown that, with blood losses of 500 cc. or more at operation, restoration by hematopoiesis is slow for at least 10 days after the loss, and when there are large deficits, restoration may be incomplete for as long as 2 months after operation.

If the total plasma volume and total plasma protein is normal with a deficit of the red cell volume, replacement by means of red cells in suspension may be desirable, especially in cases in which overloading of the circulation is a consideration. In the absence of

deficits in hemoglobin and erythrocytes, but with depleted plasma and proteins, restoration of the latter is accomplished by plasma infusions or by human serum albumin. With deficiencies of all elements of the circulation, blood volume compensation by means of whole blood or whole blood and plasma is recommended.

The circulating protein deficit is an index of the body protein deficit, the latter being 30 times the former. It further indicates disturbance in protein metabolism from inadequate intake, poor absorption, or faulty utilization. The ill effects of reduced circulating protein have been known for many years,[20] and calculation of such deficits by the methods outlined gives warning that edema of anastomotic stomas, delayed wound healing, and dehiscence, for example, may occur.

Often it is impossible to make quantitative replacement in the preoperative period and complete restitution may not be necessary. If time or disease does not allow adequate oral replacement, 35 to 40 grams of human salt-poor albumin may be added to each 1,000 cc. of whole blood.

The hemoglobin deficit is more important than the cell volume deficit because the oxygen-carrying capacity of the blood is directly proportional to the amount of circulating hemoglobin. Replacement should be made on the basis of the hemoglobin deficit and not on the basis of the cell deficit. Cell volume per kilogram does not disclose severe hemoglobin loss although there is a definite parallelism. When the hemoglobin or cell deficit is disproportionately severe, administration of a suspension of erythrocytes is better than administration of whole blood.

To estimate the amount of whole blood needed for replacement, the total circulating hemoglobin deficit in grams is divided by the standard hemoglobin concentration in grams per 100 cc. of blood; the result of this multiplied by 100 will give the cubic centimeters of whole blood needed to replace the deficit.

SUMMARY

A method has been presented which will more accurately estimate the necessary blood volume replacement in the surgical patient in an attempt to prevent shock or treat it more adequately if it should occur.

Brookhollow Dr.
Route 5

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PHYSICIAN AND PATIENT

The physician enjoys unique privileges and opportunities; on the other hand, he is burdened with special responsibilities and obligations. The law requires that, if he presumes to take professional care of a patient, he must possess and exercise that degree of skill and care that is commonly possessed and exercised by other reputable practitioners in the locality in caring for similar cases. The patient expects the physician to be a dedicated person—gentle and kind, always available, inexhaustibly patient, everlastingly resourceful, and possessed of endless charity.

Both law and ethics join in exhorting the physician to put the patient's interest above all else, in requiring him to hold the patient's communications in confidence and to act toward the patient with the utmost good faith at all times. Ethics is derived from the Greek *ethos*, and, by extension, as it is used, it means conforming to professional standards of conduct. To such a standard the physician is bound. It is in his personal concern for his patient that the physician differs from the tradesman or the pure scientist. The secret of his successful care of his patient lies in his caring for the patient.—Louis Regan, M. D., *The Journal of the American Medical Association*, May 28, 1955.

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Succinylcholine Chloride in Combined Anesthesia

GLENN F. GORDON, D.O.
Glendale, Calif.

The introduction of curare into the field of clinical anesthesia was a welcome addition to the armamentarium of the anesthetist. With its use the patient arrived in the operating room in a state of greater relaxation which was not dependent upon depression alone. Each subsequent successor to curare has constituted an attempt to provide the same muscular relaxation without the undesirable effects of curare. These undesirable properties are recognized as (1) prolonged and cumulative action, (2) histamine liberation and ganglionic blocking demonstrated as bronchiolar spasm, excessive salivation, and hypotension, and (3) potentiation of the curariform property of ether.

In spite of these hazards, curare was found preferable to the depressive doses of other agents required to produce similar effects. It is probable that the full extent of curarization was frequently avoided in order to obviate the hazards.

Succinylcholine chloride presents evidence of having met the requisites of curare with elimination of the undesirable properties. With the wave of enthusiasm which accompanied the early use of this relaxant, it was difficult to be thoroughly objective about it. This lack of objectivity is well illustrated by one resident's remark, "This makes a good anesthetist out of a poor one."

COMPARISON OF CURARE AND SUCCINYLCHOLINE CHLORIDE

Although both exert their action at the myoneural junction, there are pharmacologic differences between curare and succinylcholine. The curare series elevates the threshold of acetylcholine, thereby preventing depolarization. Succinylcholine acts with a depolarizing property on the end-plates. Succinylcholine is hydrolyzed by plasma-borne cholinesterase to the two inert substances, succinic acid and choline; rapid hydrolysis accounts for the brief duration of action. Curare is eliminated by the kidneys with little alteration. There appears to be no ganglionic blockade or histamine liberation with succinylcholine, nor is there the ether potentiation noted with curare.

Clinically, succinylcholine chloride exhibits (1) brief and noncumulative action; (2) absence of bronchiolar spasm; (3) less salivation; and (4) reasonable control of action, maximal relaxation, rapid onset, and more rapid recovery than curare.

No chemical antidote comparable to the curare antagonists has been provided for succinylcholine, but there seems little need for one. Discontinuation of its use allows rapid recovery, and, if necessary, whole blood transfusion would provide additional enzyme for its destruction.

PROCEDURE

I have anesthetized 489 patients for whom succinylcholine had been the only relaxant. The only criterion for selection of cases was that general anesthesia and a muscle relaxant were to be employed to produce muscular relaxation and allow endotracheal intubation, and control of respiration. In all instances, the continuous infusion technic was employed; a 0.1 per cent solution in 5 per cent dextrose in water was extemporaneously prepared and was not used after 8 hours. All types of surgical procedures were accepted wherein the general indications held good.

The premedication generally employed was sodium phenobarbital 2 hours preoperatively and morphine and atropine 1 hour before induction. The procedure followed was induction with Pentothal sodium, 2.5 per cent, in sufficient dosage to permit application of a face mask, followed by cyclopropane not exceeding 20 per cent, and oxygen mixture, or nitrous oxide, in variable concentrations. When the rate and depth of respirations were indicative of stability in the second plane, the infusion was begun through a 22-gauge needle. The rate of the drip was not measured in drops per minute but was gradually increased until the depth of respiration was impaired. For purposes of intubation, near, or actual, apnea was employed and the flow reduced or completely discontinued during the procedure. Respirations were assisted at a rapid rate with high oxygen concentration prior to the production of apnea.

During the surgical procedures the flow was regulated by the demands of the case. For example, during the crucial portions of the operation in biliary procedures and during surgical closure of the bronchi and vessels in thoracic operations, apnea was produced and respiration controlled.

The patients in the series ranged in age from 10 to 83 years. The average number of poor-risk cases was included. The length of anesthesia ranged from 20 minutes for strabotomy to 6 hours and 50 minutes for esophagectomy. Total dosage of succinylcholine ranged from 40 mg. to 1,500 mg. Products used were Anectine and Quelicin interchangeably. Of the series, 204 patients were managed by endotracheal technics.

OBSERVATIONS

In general, exceptional tolerance and consistent response were found in these patients. Muscular relaxation was excellent and fully comparable with that of spinal anesthesia. With greater relaxation, when cyclopropane was employed, it was found necessary to maintain assisted respiration in order to avoid the development of the characteristic cardiac irregularities associated with hypoxia and carbon dioxide retention. With submaximal relaxation, nitrous oxide was employed, using the high flow method. There were no complications.

With succinylcholine chloride the ease and facility of intubation were found to be greatly enhanced and almost entirely free of complicating spasm and cyanosis. Reaction to the presence of the tube was readily controlled by management of the flow of solution. There was no concern over prolongation of apnea, when it was produced for surgical facility and respiration was controlled. Recovery was rapid and uneventful.

In one case, a 59-year-old female was anesthetized for removal of a nasal polyp. Previously, numerous unsuccessful attempts at topical anesthesia had been made. Pentothal sodium, 0.2 gram, was injected slowly, and the anesthetic machine was attached with the face mask. Preparatory to intubation, succinylcholine was introduced at a moderate rate and the respiration

TABLE I—SUMMARY OF OPERATIONS

Type of Operation		Total No. of Cases
Upper abdomen		45
Including		
Gastrectomy	12	
Biliary tract	31	
Gynecologic		
(Including operations on the perineum)		140
Abdominal wall		33
Lower abdomen		108
E.E.N.T.		41
Including		
Mastoidectomy	1	
Caldwell-Luc	2	
Open reduction malar	1	
Strabotomy	3	
Tonsillectomy (adult)	32	
Major intestinal		28
Including		
Abdominoperineal resection	5	
Transanal pull-through	1	
Urologic		
Including		
Nephropexy	6	
Nephrectomy	1	
Prostatectomy	1	
Thyroidectomy		9
Mastectomy		7
Cesarean section		6
Thorax		19
Including		
Decortication	2	
Esophagectomy	1	
Diaphragmatic hernia	5	
Commissurotomy	3	
Lobectomy	4	
Pneumonectomy	4	
Rectal		13
Neurosurgery		5
Including		
Gasserian ganglionectomy	1	
Craniotomy	1	
Rhizotomy	1	
Sympathectomy	1	
Orthopedic surgery		19
Including		
Laminectomy	7	
Laminectomy with fusion	5	
Spinal fusion	1	
Hip reconstruction	2	
Peripheral vascular disease surgery		1
Miscellaneous		6

observed. Obstructed efforts were present with the absence of any exchange. Laryngoscopy revealed abducted cords with a relaxed, patent larynx through which a large catheter (No. 9 Portex) was inserted. Positive pressure ventilation re-established spontaneous respiration. This was the only case in which bronchospasm was demonstrated. It may well be that Pentothal was the inciting factor.

The succession of effects was demonstrated by a case not included in this series. A 46-year-old male was hospitalized because of inability to open his jaws 8 days after a dental extraction. Examination of the mouth was impossible. Succinylcholine chloride infusion, 0.1 per cent, was employed with the cooperation of the patient. Following the infusion of 70 mg., his mouth could be opened approximately 2 cm.

He reported that the first effect was disturbance of vision, which was followed by loss of facial function, loss of function of the extremities, and finally respiratory disturbance. This is the only case in which the agent was employed in a conscious patient.

The following findings related to the pharmacology of the drug were observed: (1) Nine patients with hemoconcentration were presented for elective operations. Each of these required more than the usual dosage in respect to the duration and nature of the operative procedure. (2) The total dosage varied most constantly with the length of operation because of the short action of the drug. (3) Patients with anemia demonstrated less tolerance than usual, and in them the action of the drug was more prolonged than normal. Concomitant blood transfusion is advised in the presence of pre-existing anemia.

The much-discussed muscular fasciculations were not observed among these cases, probably because of the barbiturate (Pentothal) used and the slow administration of succinylcholine.

Endotracheal intubation was not employed in all cases and is not imperative when submaximal relaxation is adequate. However, the equipment should be immediately available should the necessity arise. Succinylcholine is not used without a provisional closed system in operation. With few exceptions (14 cases), surgical procedures on the female pelvis were conducted without endotracheal intubation by allowing spontaneous respiration and submaximal relaxation which was entirely adequate.

Respiration was not generally changed in rate but with progressive effect the depth was consistently decreased. Following the initial response of the individual to succinylcholine, the responses thereafter are similar and readily predictable.

Tachycardia and elevation in blood pressure occur in some cases with diminished respiratory excursion, and both are related to carbon dioxide retention. This can be prevented or corrected simply by augmenting the respiratory exchange by manual means or by means that insure exhaust of the carbon dioxide (high flow of gases).

Salivation occurred in a few of the cases and was most notable when intubation was attempted before the patient was conditioned properly; in these cases more than one attempt was necessary for completion of the procedure.

In addition to using succinylcholine chloride in my private practice, the drug has replaced curare in our resident training in the hospital. It is felt that with employment of a 0.1 per cent solution in the infusion technic, there is a wider margin of safety, particularly after a period during which the basic agents are used and the trainee familiarizes himself with their properties in the technics commonly employed.

CONCLUSION

Succinylcholine chloride is, at present, the best muscle relaxant available. Rapid onset, minimal side effects, and rapid recovery from the maximal effect permit an extreme degree of control and a broad margin of safety. Once a given patient has been evaluated and response established, the rate of infusion must be balanced between the depth of respiration and the degree of muscular relaxation. When these are incompatible, respiration is maintained either by assistance or control.

Management could be regulated best with use of the 0.1 per cent solution. The introduction was without muscular fasciculation, and the degree of myoneural

effect was readily altered by minor adjustments in the rate of flow of the solution.

Recognition of the mode of destruction of the drug directs attention to the condition of serum deficiency and also the importance of serum replacement in providing sufficient cholinesterase for hydrolysis of succinylcholine chloride.

1385 E. Colorado Blvd.

Interrelationship of Cardiac and Respiratory Physiology—A Possible Explanation of Cardiac Arrest

MAXWELL R. BROTHERS, D.O.
Los Angeles

It is not the purpose of this paper to solve the problems of cardiac arrest, but rather to review the basic mechanisms of cardiac physiology in an attempt to interrelate their dependency upon the basic physiologic mechanisms of respiration.

Cardiac muscle may be described as a continuous multinucleated sheet over which impulses may spread in any direction without interruption. When it is stimulated it obeys the all or none law, and its reaction to a given stimulus depends upon the conditions existing at the time. It has a relatively long refractory period which accounts for the compensatory pause following premature systoles, helps keep the heart under the control of the sinoatrial node, and prevents tetanus. This refractory period may be shortened by an increase in temperature and acceleration of the heart and may be lengthened by drugs such as quinidine.[1]

In the metabolism of cardiac muscle, lactic acid is preferred to glucose, lactic acid being oxidized by the heart muscle for the restoration of the glycogen stores and the resynthesis of phosphocreatine and adenosine triphosphate for energy in muscle contraction. Where glucose, glycogen, and lactate are burned by the heart, glucose is used mainly to restore the glycogen stores. These stores may be rapidly depleted by the administration of epinephrine. In the absence of an adequate amount of lactic acid, the heart may derive its sole energy requirements from noncarbohydrate material such as fat, glycogen being used only as an emergency fuel in heavy work. However, lactic acid, glucose and fat, when available, may all serve as sources of energy.[1]

Muscle is composed of from 18 to 20 per cent protein and between 75 and 80 per cent water. The remainder is made up of various minerals and a large number of organic compounds. The contractile elements of muscle protein are myosin and actin, which account for 60 per cent and 12 per cent respectively of the total protein. Potassium, magnesium, sodium, and calcium play an important role, and their proper concentrations are necessary to maintain the excitability and contractility of cardiac muscle.[1]

The coefficient of oxygen utilization of cardiac muscle is very high. It is estimated that approximately 5 per cent of the total oxygen consumed by the body in the resting state is utilized by the heart. In severe exercise, the heart may consume as much oxygen as the entire body under basal conditions, and the content of the coronary venous blood may be reduced to practically zero.

The action of epinephrine accelerates the heart and increases the oxygen consumption by 20 to 40 per cent and carbon dioxide production from 30 to 50 per cent. With a 50 per cent reduction of arterial oxygen, there is diminution in the size of the T wave on the electrocardiogram, and it may become flattened or inverted if hypoxemia persists. The heart can stand but a very small oxygen debt and ceases to function after 4 or 5 minutes in the absence of oxygen or when the debt amounts to 0.06 cc. per gram of tissue. High oxygen tensions reduce slightly and very low tensions increase the output of the heart.

Heart muscle has a very poor buffering power, and when the hydrogen ion concentration falls to around 7, extrasystoles, heart block, or other irregularities occur and are followed by cardiac arrest. The presence of acids favors relaxation of cardiac muscle and decreases conductivity through the atrioventricular bundle. Arrest occurs in diastole. Alkalis, on the other hand, prolong systole, shorten diastole, and increase conductivity through the atrioventricular bundle.

Carbon dioxide exerts an effect upon the cardiac and vasomotor centers and directly upon the cardiac musculature. It dilates the capillaries and small veins and assures a more generous return to the right heart. By its relaxing effect upon the cardiac muscle fiber, it enhances the extensibility during diastole, favoring an increased filling of the chambers and resulting in an increased stroke output. It excites the vasoconstrictor center, thus constricting the arterioles, stimulates the cardioinhibitory center, and depresses the activity of the sinoatrial node. Thus the cardiac output is increasd, and there is an elevation of the blood pressure with a slowing of the rate. The activity of junctional tissue is suppressed with high carbon dioxide concentrations, and when there is sufficient increase to lower hydrogen ion concentration to around 6.5, complete heart block occurs. With an increase of the carbon dioxide tension over a prolonged period, there is weakening of the heart beat, the development of irregular rhythms, and a consequent fall in blood pressure.

With a carbon dioxide deficit, there is an increased hydrogen ion concentration, an increase in the conduction of the atrioventricular bundle, initiation of impulses in the sinoatrial node, and a reduction in tone of the cardioinhibitory center. Diastole is incomplete, and there is an increased tone in the capillaries and small veins with a drop in venous pressure. Consequently, there is a decreased return of blood to the heart with insufficient filling of its chambers, and the cardiac output is reduced. With reduction in diastole, the cardiac rate increases to a point where it may be in almost continuous contraction. There is a fall in blood pressure, and death occurs from circulatory failure.

Since oxygenation of arterial blood and the elimination of carbon dioxide from venous blood in its return

from the tissues requires adequate ventilation, good respiratory function is necessary to insure cardiac activity. This is especially true with the diseased myocardium where the demands for oxygen may be high, and the mechanisms for supplying the oxygenated blood are impaired or have not compensated for the increased demand.

The air of the anatomic dead space, amounting to approximately 150 cc., does not enter into active ventilation, merely being shuttled back and forth from the outside air to the anatomic dead space.[1] Active ventilation is dependent upon mixing an excess of this dead space air with supplemental and residual airs in order to increase the oxygen tension and reduce the carbon dioxide tension of alveolar air. The gaseous exchanges between inspired air and alveolar air, between alveolar air and the blood in the pulmonary capillaries, and between the blood in the tissue capillaries and the tissues are governed by physical laws as postulated in the kinetic theory of the exchanges of gases.

The interchange of gases and transportation via the blood stream is dependent upon many factors. There must be an adequate amount of air, containing oxygen in excess of that in the anatomic dead space, to assure the maintenance of effective oxygen tension and the elimination of carbon dioxide. There must be adequate excursion to insure this interchange in the respiratory tree.

The flow of air into and from the lung depends entirely upon the changes in capacity of the thoracic cavity. The lungs play a purely passive role. During the act of inspiration, all parts of the lung do not expand equally or directly. Those that expand to the greatest extent are expanded directly and are in contact with the mobile portions of the chest such as the sternum, the ribs, and the diaphragm. The remainder of the lung expands indirectly and depends to a large extent upon the mobility of the lung tissue. During inspiration there is elongation of the bronchial tree, the bronchioles and smaller bronchi dilate, and the areas of the lung that are expanded indirectly become active in the process of respiration. Therefore, any pathologic entity which fixes the root of the lungs interferes with their normal descent during inspiration and inhibits the expansion of the alveoli that are affected indirectly. During expiration, with reduction in the size of the thoracic cavity, the highly elastic bronchial tree recoils to its previous length, and the lung root ascends, aiding the elimination of tidal air.

Under ordinary physiologic conditions during quiet respiration, all of the alveoli are not equally expanded; consequently the blood supplying the unexpanded alveoli will not be completely saturated with oxygen. The blood in the alveoli that are completely expanded will be saturated, and the admixture of the blood will reduce the total oxygen saturation. In association there is an admixture of the blood in the bronchial and pulmonary vessels through their anastomoses in the respiratory bronchioles; thus the arterial oxygen saturation is approximately 9 mm., less than the oxygen in the well-ventilated alveoli. Oxygen saturation may be further diminished during surgical procedures with abnormal positioning which restricts respiratory excursions. This is especially true in thoracic surgery with the patient lying on his side with the opposite lung collapsed. Oxygen deficiency is further exaggerated by blood stasis in the dependent tissues.[2] Overventilation cannot compensate for this oxygen deficit to any significant extent.

With increased activity of any tissue there is an increase in oxygen consumption by several times that of the resting value. Increasing the oxygen supply to the tissue above its requirement does not increase its usage, the tissue taking what oxygen it demands for the moment and no more, which precludes the possibility of storage of this vital gas for future use. The only way the oxygen supply to the tissues is met is by increasing the total blood flow or by increasing the coefficient of oxygen utilization.

The oxygen saturation in the lungs depends upon an adequate pressure head of 80 mm. of mercury for maximal saturation. In a reduced oxygen pressure, as occurs in the tissues, there is dissociation of the oxyhemoglobin to reduced hemoglobin. An increased temperature, or a shift of the hydrogen ion concentration of the blood toward the acid side, reduces the affinity of hemoglobin for oxygen. Thus in the tissues the carbon dioxide, lactic acid, and increased temperature favor dissociation of the oxyhemoglobin at the lower oxygen pressure. However, these agencies have little effect on the oxygen uptake of hemoglobin at the higher oxygen tensions necessary for maximal saturation. On the other hand, alkalis and a fall in temperature have a reverse effect. The changes in the hydrogen ion concentration of the plasma are minimized by means of the chloride shift between the red blood cells and the plasma and by the buffering action of plasma proteins.

Whereas an intact nervous system is unnecessary for the maintenance of cardiac activity, it plays an important part in the regulation of the rate and the maintenance of the normal rhythm. With an intact nervous system the heart is continually under the influence of impulses originating in the cardiac centers and transmitted through the vagus and accelator nerves to the heart, the cardiac centers receiving their stimuli or impulses from various portions of the body, the heart included.

The vagus is cardioinhibitory, and its fibers pass to the auricle but for the most part terminate in the sinoatrial and atrioventricular nodes. None of the vagal fibers terminate in the ventricular myocardium. The right vagus goes principally to the sinoatrial node with small filaments to the auricular muscle and the atrioventricular node, the left vagus to the atrioventricular node with small filaments to the sinoatrial node and auricle. Stimulation of the vagus causes prolonged slowing or complete stoppage of the heart with a consequent fall in blood pressure by the prolongation of diastole. In this respect it resembles the action of an excess of potassium and of acids on heart muscle. However, when the heart stops by virtue of vagal excitation, the ventricles but, as a rule, not the auricles will start to beat again after a time. This is termed "cardiac escape" and is probably due to the activation of an ectopic focus lower in the connective tissue which is enhanced by the action of epinephrine and the sympathetic nerves. The vagus thus exerts a continuous restraint upon the heart action which may be annulled by the use of atropine. This vagal action is apparently reflex in nature and dependent upon impulses coming to the vagal centers along the sinus and aortic nerves.[1]

The accelerator nerves arise from cells situated in the lateral horn from the first to the fifth thoracic segments of the spinal cord and these cells constitute a

spinal cardioaccelerator center. The preganglionic fibers ascend in the gangliated cord of the sympathetic to connect with cells of the superior, middle, and inferior cervical ganglia. The postganglionic fibers from these cervical ganglia form the superior, middle, and inferior cardiac nerves. In addition, there are accelerator fibers passing directly from the sympathetic chain to the heart as far down as the fourth and fifth thoracic ganglia. As with the vagus, the fibers on the right terminate in the sinoatrial node and on the left in the atrioventricular node and bundle, with fibers to the ventricular muscle. The superior cardiac nerve is distributed to the large arteries. The efferent fibers to the heart are contained mainly in the middle cardiac nerve, while the inferior cardiac nerve is mainly afferent. Stimulation of the accelerators causes an increase in the heart rate of both the auricles and the ventricles with an increase in the force of contractions. This is accomplished through the shortening of both systole and diastole, with systole being curtailed to a greater degree. Excessive stimulation may excite the ventricles into fibrillation.[1]

The changes in heart rate are brought about by the reciprocal action of both the cardioinhibitory and the cardioaccelerator centers, the cardioinhibitory center exerting a greater influence than the accelerator center. These centers exert their influence primarily through reflex pathways, the afferent stimuli arising from all parts of the body, including the heart, and their effects on the heart being mediated through the cardiac centers. The stimulation of the oculocardiac reflex, the carotid sinus reflex, tugging on the intestines, and inhalation of irritating vapors may cause reflex inhibition of the heart. The excitation of the central ends of peripheral nerves such as the sciatic will cause acceleration that is thought to be due to an overflow of impulses from the cerebral centers to the cardiac centers. With an increase in blood pressure there is slowing of the heart rate that is dependent upon reflex afferent vagal fibers ending in the aortic arch, the heart, and the carotid sinus.

When there is an increase in the venous pressure of the blood entering the right heart, there is a reflex increase in the heart rate. This is known as the Bainbridge reflex, and, by this reflex, the heart compensates for an increase in venous return to the heart. It is dependent upon afferent vagal stimulation through nerves terminating beneath the endocardium and the walls of the great veins near their entrance to the auricles.

Patterson and Starling[3] demonstrated in the dog's heart that the output of the heart was directly proportionate to the diastolic filling up to an optimal degree of dilatation of the cardiac chambers, and this was independent of reflex pathways. Beyond the optimal filling there was a falling off of the output as the heart became excessively dilated and failed to eject as much blood as it received. The flow of the venous blood to the heart is increased during inspiration by the increase in the effective venous pressure due to the negative pressure within the chest. The descent of the diaphragm also increases the intra-abdominal pressure and the pressure on the inferior vena cava, augmenting the flow of blood toward the heart. The result is an increased filling of the right auricle with an increase in the stroke volume output, according to Starling's efficiency curve, and acceleration of the rate by stimulation of the Bainbridge reflex.

Within physiologic ranges, inflation and deflation of the lungs will cause, respectively, acceleration and slowing of the heart. This reflex is brought about through afferent pulmonary stimuli causing a depression in tone of the vagus center (that is, cardioinhibitory) with a consequent release of vagal influence and an increase in heart rate. The afferent impulses apparently originate in the stretch receptors in the visceral pleura or lung immediately subjacent to it. These receptors adapt rapidly, and, if the inflation is protracted, there is secondary slowing of the heart. With extreme inflation of the lungs, cardiac slowing rather than acceleration occurs. The slowing of the heart caused by deflation of the lungs is accompanied by depression of atrioventricular conduction; thus, in a heart in which there is already some delay in conduction over the bundle, complete heart block may be caused by rapid deflation.[1]

The acceleration of the heart in emotional states and during muscular exercise is thought to be initiated by an overflow of stimuli from the cerebral cortex, resulting in vagal depression. The continued acceleration in these states is brought about through the Bainbridge reflex and the liberation of epinephrine.

According to the theory of the humoral transmission of impulses, acetylcholine is the apparent active principle liberated by vagal stimulation. This substance is destroyed by alkali or cholinesterase but is stable in acid media. Thus the accumulation of lactic acid and carbon dioxide favors the prolongation of the action of acetylcholine, and the vagal effect is enhanced. This is a possible mechanism for the final production of cardiac arrest by vagal stimulation when there is an excess of carbon dioxide and lactic acid in the blood stream and fluids bathing the heart.

Respiration is directly under the control of the respiratory center in the brain which discharges impulses over the motor neurons of the phrenic and thoracic nerves in the cervical (3, 4, and 5) and the upper thoracic segments (2 to 6) of the spinal cord. Spontaneous respiratory activity is apparently dependent upon the inspiratory center. The expiratory center plays an inhibitory role to interrupt intermittently the inspiratory discharge, resulting in expiration which in quiet breathing is probably entirely a passive movement. In ordinary breathing, overdistention of the lungs at the end of an inspiration of the usual duration is prevented by the Hering-Breuer reflex and is mediated through the afferent fibers of the pulmonary vagi.[1]

Stimulation of almost any afferent nerve may bring about reflex changes in respiration. This may be demonstrated by the cessation of respiration during the act of swallowing and the stimulation of the nasal mucosa by irritating gases.

Chemical stimulation of the respiratory center is carried out through the action of carbon dioxide under ordinary physiologic conditions. The cells of the respiratory center are different from the rest of the central nervous system in that they are stimulated by the carbon dioxide tension and hydrogen ion concentration. They are like the cells of the remainder of the central nervous system in that they are depressed by oxygen lack. Carbon dioxide increases primarily the depth of respiration and secondarily the rate. An increase of 0.2 per cent in alveolar air is sufficient to increase pulmonary ventilation substantially.

In the carotid and aortic areas there are reflexes containing both pressoreceptors (which respond to mechanical stimuli) and chemoreceptors (which respond to chemical stimuli). The pressoreceptors, which tend to

inhibit respiration, serve no significant physiologic respiratory function in man except, possibly, in strenuous exercise. The chemoreceptors are stimulated by oxygen lack, but not until the oxygen tension is below 92 per cent saturation, and they increase the rate and depth of breathing. The chemoreceptors are less sensitive to carbon dioxide, and it is felt that they do not play an important role in respiration under ordinary physiologic conditions. Hypoxia is apparently an ineffective stimulus to the respiratory center and, in addition, may depress and cause failure of the central neurons. The chemoreceptors are highly resistant to hypoxia and will continue to exert their influence on the respiratory center which, otherwise in anoxic states, would be unresponsive to the emergency. The chemoreceptors are relatively insensitive to change in the hydrogen ion concentration and are thought to be stimulated by the acid products of their own metabolism which may be greatly altered by a deficient oxygen supply and an accumulation of lactic acid.[1]

The chemical physiology of muscular contraction is divided into two phases, aerobic and anaerobic. In the anaerobic phase the muscle may continue to contract for 4 or 5 minutes without an adequate supply of oxygen necessary for its recuperation; however, forceful contractions will diminish until the muscle is fatigued. During this time, there is an increase in the lactic acid concentration and a decrease in the glycogen store. Some carbon dioxide is produced in this phase because of the action of lactic acid on bicarbonate in the muscle fluids. For the most part, carbon dioxide is not formed until the oxidative phase occurs and glycogen stores are replenished. In the anaerobic phase of muscle contraction there is a cleavage of the terminal phosphate from adenosine triphosphate, which is catalyzed by adenosine triphosphatase, and yields phosphoric acid, adenosine diphosphate, and 12,000 calories of energy for contraction.[1]

Simultaneously the released phosphate aids in the phosphorylation of glycogen in which adenylic acid acts as a coenzyme. Adenosine triphosphate is resynthesized through phosphate donated by phosphocreatine, giving off creatine and 10,000 calories of energy for the resynthesis of adenosine triphosphate. The breakdown of glycogen to lactic acid provides energy for resynthesizing phosphocreatine and later, as contraction proceeds, for resynthesizing adenosine triphosphate, in which phosphate-bond energy is stored. The energy for the resynthesis of phosphocreatine and adenosine triphosphate from the reduction of glycogen to lactic acid is 29,000 calories. In the aerobic phase that occurs after active contraction, oxygen is admitted and one-fifth of the lactic acid produced is oxidized, which yields 325,000 calories of energy for the resynthesis of the remaining four-fifths to glycogen. If the oxygen supply is adequate, little or no lactic acid is formed; however, in the absence of oxygen, there is an accumulation of lactic acid which slows and then arrests the glycogen-lactic acid breakdown. The result is that the resynthesis of phosphocreatine and adenosine triphosphate is prevented. Thus, in the absence of an adequate supply of oxygen, glycogen stores are depleted. There is an increased secretion of urinary phosphorus, a gradual decrease of the phosphocreatine available for the resynthesis of adenosine triphosphate, and the energy stores for muscle contraction are exhausted.[1]

Cardiac arrest, although not a frequent problem, is the most serious of operating room emergencies. The sequence of events leading to this complication has been discussed at great length and many etiologic theories postulated. A very busy surgical center such as the Lahey Clinic anticipates approximately two cases of cardiac arrest each year.[4]

Vagal reflexes were the first etiologic mechanism suggested in the production of cardiac arrest and are still believed to be an important cause.[4-8] The studies of Reed and Brace[8] support such reflex mechanisms in the production of cardiac arrhythmias and arrests during anesthesia. However, in recent years the trend has been away from the vagal reflex theory toward one in which some other factor is associated in this complication. The various anesthetic agents and drugs have been incriminated because of their histotoxic effect, overdosage, or sensitization of the myocardium, vital centers, and reflex pathways. It has been advocated that the combination of these agents with vagal reflex phenomena is sufficient to account for many cases of cardiac arrest.[4-7,9] That an overdosage of an anesthetic agent can readily cause death through its histotoxic effect is not questioned; however, such a mechanism would seem unlikely in the hands of a well-trained anesthetist in present-day practice.

It is well known that the heart has inherent qualities for trying to maintain its activity even though impulse transmission is entirely blocked through the bundle of His and its branches (that is, cardiac escape). Sloan[10] attempted to produce cardiac arrest by direct vagal stimulation with a strong faradic current at the hilum of the lung. He was unable to do this in a well-known oxygenated animal; however, he was successful with extreme degrees of hypoxia. When arrest occurred it was with extreme rapidity. His conclusion was that vagal reflexes arising from intrathoracic operations are of little importance in well-oxygenated patients but may play a minor role in augmenting an existing oxygen deficiency to produce cardiac arrest.

While it is possible to have an oxygen excess with a carbon dioxide excess in arterial blood, as may occur in emphysema and the administration of oxygen[2] or during anesthesia when the patient is being given 100 per cent oxygen,[11] it is unlikely that this would occur under usual anesthetic conditions. Therefore, it would seem more likely that hypoxia and hypercapnea would be the combination of factors leading to cardiac arrest. That this combination, that is, hypoxia and hypercapnea, may lead to arrhythmias, overdistention of the heart, and, finally, cardiac arrest, has been observed in animals.[9,12] With improvement of ventilation before arrest occurs, the arrhythmias subside, the tone of the heart is improved, and there is a return to homeostasis. However, if carried to the point of cardiac arrest, the mere admission of oxygen and ventilation of the animal will not restore the heart activity, but cardiac massage will be required.[9,12] The work of Denson and Joseph[13] indicates that this abnormal state of ventilation is a possible mechanism of cardiac arrest in the human during anesthesia, and that cardiac arrest is not due to the introduction of intratracheal catheters or vagal reflexes.

That patients can tolerate quite high concentrations of carbon dioxide is pointed out by several investigators;[2,11] however, a sudden loss of carbon dioxide with a rapid rise in the hydrogen ion concentration of the blood may be a mechanism leading to circulatory collapse.[2,11] It would seem, then, that perhaps the prime reason for cardiac arrest during anes-

thesia is the gradual progressive hypoxia and hypercapnea, which may be increased by the effects of anesthetic agents and reflex vagal stimulation, or the sudden shift of carbon dioxide and hydrogen ion concentration in overventilation with pure oxygen.

Lovejoy,[2] in his studies on severe emphysematous patients in the Drinker respirator, has demonstrated that adequate ventilation can be maintained and sustain the individual under severe conditions, providing proper therapy is instituted and a patent airway is present. In anesthesia, ventilation may be maintained by sufficient compression of the rebreathing bag, providing an adequate airway is maintained.[14] However, the ability to estimate how much respiratory exchange occurs is extremely variable with different anesthetists; therefore, it behooves the anesthetist periodically to compare his clinical judgment and abilities with some standard in order to insure adequate ventilation of the anesthetized patient during periods of apnea or diminished respiratory excursion.

With inadequate ventilation, there will be an increase in alveolar carbon dioxide and a decrease in oxygen pressure resulting in increased carbon dioxide retention and decreased oxygen saturation of the blood. The carbon dioxide backs up in the tissue fluids and at first increases respiratory excursion, dilating the capillaries and small veins and thereby increasing the return of blood to the right heart. This is enhanced by an increase in respiratory rate. There is a compensatory increase in the blood pressure in response to the effect of carbon dioxide excess and oxygen deficit on the vasoconstrictor centers and an increase in the stroke volume due to increased filling of the chambers (Starling's efficiency curve) and oxygen deficit.

Through the Bainbridge reflex and oxygen deficit there is an acceleration of the rate which increases the cardiac minute volume output. This is directly associated with increased work of the heart and greater demand for oxygen. As the heart continues to contract in a deficit of oxygen, there is a decrease in the hydrogen ion concentration of the blood due to an increase in carbonic acid and an uncompensated bicarbonate increase. The acetylcholine activity produced by vagal stimulation is enhanced and results in slowing of the heart and production of ectopic rhythms. With prolonged carbon dioxide excess and oxygen deficit there is a weakening of the muscle fibers of the heart (obeying Starling's efficiency curve on the side of decompensation) and a resultant decrease in cardiac stroke and minute volume output and a reduction in the effective arterial pressures in the coronary arteries. Impulse transmission of the conducting tissue is impaired, ectopic rhythms, heart block, and, finally, cardiac arrest in diastole occur, with a loss of energy for muscle contraction.

If there is a sudden shift from a carbon dioxide excess to a carbon dioxide deficit by overventilation, there is an increase in the hydrogen ion concentration of the blood (that is, uncompensated alkali excess), and failure of the respiratory center to respond to the reduction in carbon dioxide. With the stimulus of hypoxia on the chemoreceptors removed by the administration of oxygen, respiratory arrest will ensue. Transmission of impulses over the conductive pathways is enhanced (carbon dioxide deficit and alkali excess), and there will be incomplete filling of the heart chamber with the resultant fall in blood pressure and increased rate until the heart is in a state of almost continuous contraction. Finally death will result from cardiac failure.

CONCLUSIONS

1. It would appear that the individual patient has physiologic mechanisms to regulate his cardiorespiratory activity and that these are interrelated and each is dependent upon the other to maintain the homeostatic state. Apparently the maintenance of this homeostatic state during anesthesia depends basically upon adequate ventilation to avoid the complication of cardiac arrest.

2. Some cases of cardiac arrest cannot be explained on the basis of this hypothesis, but they may be due to sensitization to some chemical agents such as that occurring with spinal anesthesia.

3. Because of the escape mechanism of the heart, vagal reflexes are unlikely to be the single etiologic mechanism in the production of cardiac arrest, but they may play a minor role when accompanied by hypercapnea and hypoxia.

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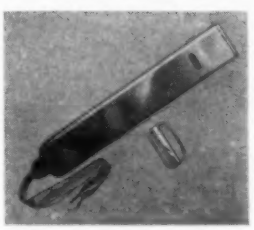

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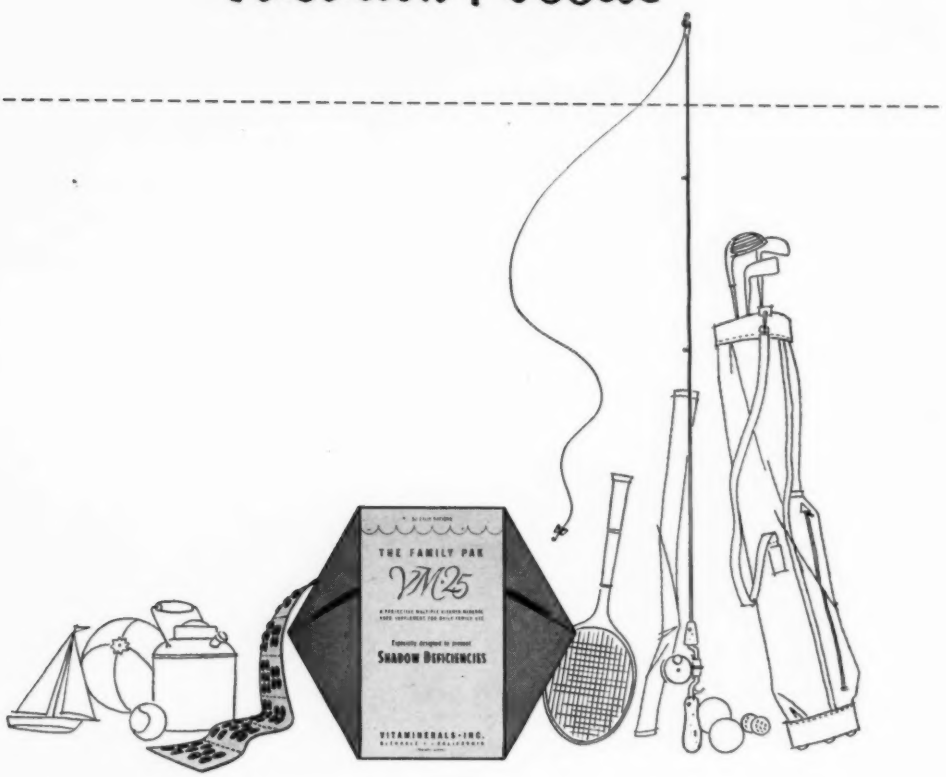

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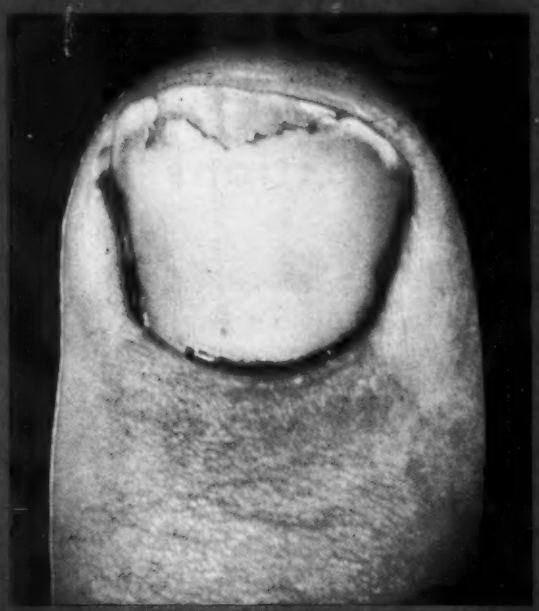

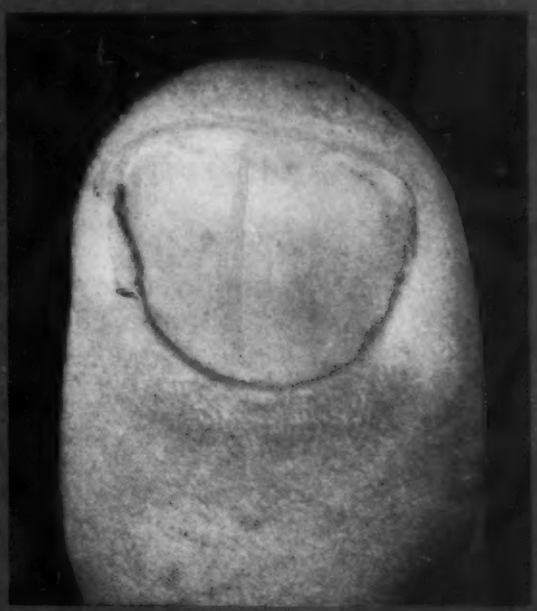

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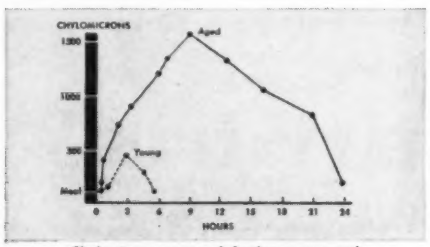
It has also been shown that (1) a high incidence of hypercoagulability and low blood heparin levels exist in patients with cardiovascular disease and atherosclerosis; (2) circulating heparin tends to decrease with age; and (3) an inverse ratio exists between the concentration of giant lipoprotein molecules and serum heparin levels.



Chylomicron curves of fasting young and old subjects after a Standard fat meal. After Becker et al: Science 110:529, 1949.

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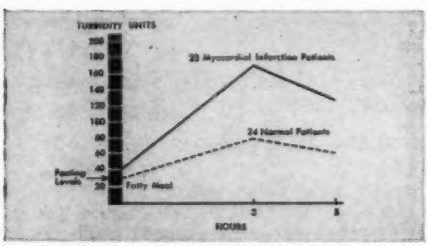

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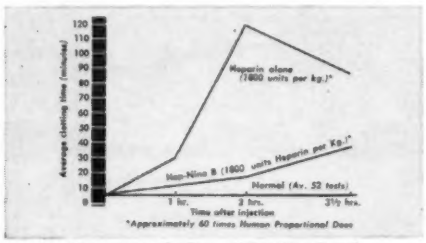

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*Read, J. T., and Obetz, R. C.: Clinical Experience with Parenteral Heparin-Lipotropic Therapy in Cardiovascular Diseases. Ohio State M. J. (In press).

Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, College of Osteopathic Physicians and Surgeons and Biltmore and Statler Hotels, Los Angeles; Convention Instruction Courses, July 5-16; Convention Teaching Sessions, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles 8.

Academy of Applied Osteopathy, annual meeting, Statler Hotel, Los Angeles, July 22-23. Program Cochairmen, Margaret H. Raffa, 5009 Central Ave., Tampa 3, Fla., and Paul K. Theobald, 1419 Broadway, Oakland 12, Calif. Secretary, Margaret W. Barnes, Box 1345, Carmel, Calif.

American College of Osteopathic Internists, annual meeting, Sheraton-Cadillac Hotel, Detroit, October 20-22. Program Chairman, Ward E. Perrin, Chicago Osteopathic Hospital, 5250 S. Ellis Ave., Chicago 15. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Neil House Hotel, Columbus, Ohio, February 6-9. Program Chairman, Richard E. Eby, 1247 N. Park Ave., Pomona, Calif. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Arthur M. Flack, 135 S. 17th St., Philadelphia 3. Executive Secretary, Orel F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Washington, D. C., October 29-November 2. Program Chairman, Karl P. B. Madsen, 460 Staten Ave., Oakland 10, Calif. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8.

American Osteopathic Academy of Physical Medicine and Rehabilitation, annual meeting, Los Angeles, July 23. Program Chairman, W. Irvin Harner, 302 W. Hadley St., Whittier, Calif. Secretary, John A. Schuck, 1739 Griffin Ave., Los Angeles 31.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, Harry J. Petri, Jr., 5 Deering St., Portland 3, Maine. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Program Chairman, A. G. Reed, 212 Pythian Bldg., Tulsa 3, Okla. Secretary, F. A. Turfler, Jr., South Bend Osteopathic Hospital, 118 S. William St., South Bend 2, Indiana.

American Osteopathic Hospital Association, annual meeting, Hotel Statler, Washington, D. C., October 30-November 2. Program Chairman, Mrs. Alixe P. Nuzum, Des Moines General Hospital, Des Moines, Iowa. Executive Secretary, Mr. R. P. Chapman, 1013 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

Canadian Osteopathic Association, annual meeting, Mount Royal Hotel, Montreal, October 27-29. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25. Secretary, Miss Joyce C. Currie, 609 Medical Arts Bldg., Montreal 25.

Hawaii, annual meeting, Reef Hotel, Honolulu, July 25-27. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13. Secretary, Joseph W. Stella, 38 Young Hotel Bldg., Honolulu 13.

Kentucky, annual meeting, Brown Hotel, Louisville, October 12-13. Program Chairman, Henry P. Lawrence, Hawesville. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland.

Maine, midyear meeting, Eastland Hotel, Portland, December 2-3. Secretary, Roswell P. Bates, 72 Main St., Orono.

Massachusetts, annual meeting, Hotel Somerset, Boston, January 21-22. Program Chairman, Everett L. Pierce, Pleasant St., East Dennis. Executive

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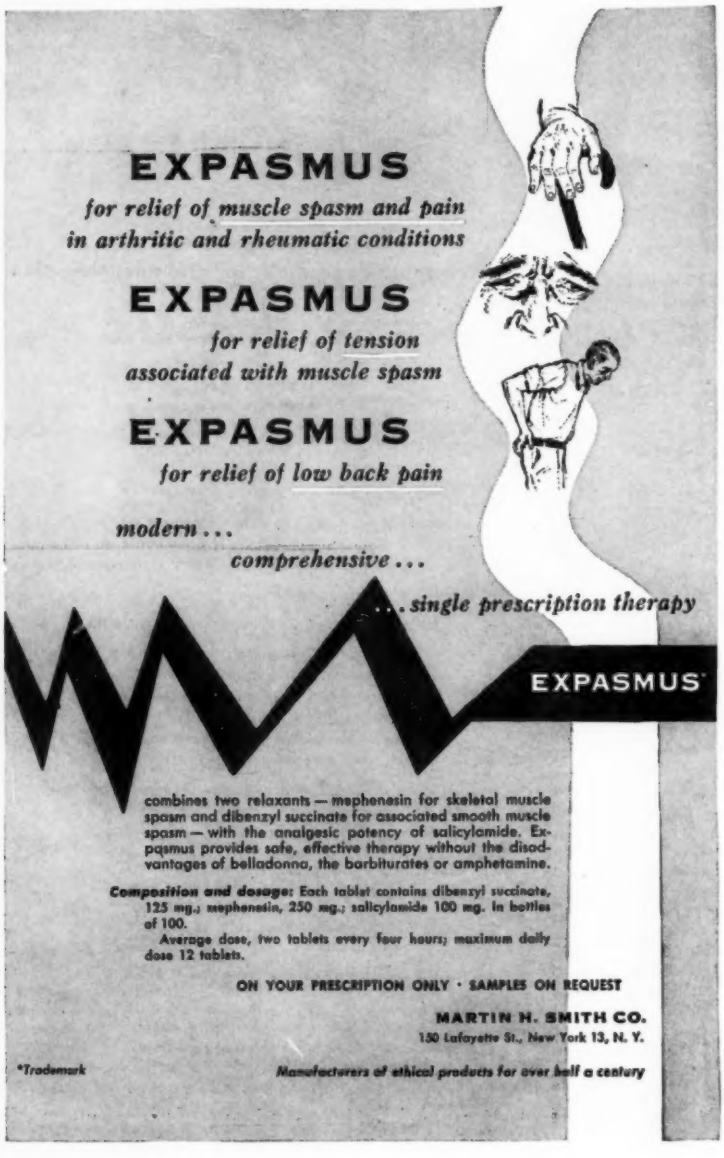
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Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 3-5. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe. Executive Secretary, Mr. Harve Lamont Smith, 81 Glendale, Highland Park 3.

Missouri, annual meeting, Continental Hotel and Municipal Auditorium, Kansas City, September 27-29. Program Chairman, J. Edward Sommers, 6504A Delmar Blvd., St. Louis 5. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City.

New Jersey, midyear meeting, Stacy-Trent Hotel, Trenton, September 18. Annual meeting, Essex House, Newark, March 10-11. Program Cochairmen for both meetings, George W. Northup, 104 S. Livingston Ave., Livingston, and Harry A. Sweeney, 25 S. Illinois Ave., Atlantic City. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside.

New York, annual meeting, Hotel Statler, New York City, October 14-15. Program Chairman, Walter Streicker, 7 Plaza St., Brooklyn 17. Secretary, Robert E. Cole, 417 S. Main St., Geneva.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 20-22. Secretary and Program Chairman, S. Dales Foster, 710 Public Service Bldg., Asheville.

Oklahoma, annual meeting, Hotel Tulsa, Tulsa, November 8-10. Program Chairman, Carl R. Samuels, S. Adair St., Box 44, Pryor. Executive Secretary, Mr. Walter L. Gray, 210-12 Braniff Bldg., Oklahoma City.

Osteopathic College of Ophthalmology and Otorhinolaryngology, annual meeting, Continental Hotel, Kansas City, Mo., July 24-27. Program Chairman, H. Mahlon Gehman, 1818 Pine St., Philadelphia 3. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15, Ohio.

Pennsylvania, annual meeting, Bellevue-Stratford Hotel, Philadelphia, September 30-October 2. Program Chairman, George S. Esayian, 113 N. Ambler St., Quakertown. Executive Secretary, Mr. George W. Thomas, 1941 Market St., Harrisburg.

Vermont, annual meeting, Long Trail Lodge, Mendon Mt., Rutland, September 24-25. Program Chairman, Hermon K. Sherburne, Jr., Mead Bldg., Rutland. Clerk, Howard I. Slocum, Battell Block, Middlebury.

Washington, midyear meeting, Washington Athletic Club, Seattle, December 3-4. Program Chairman, Erskine H. Burton, 710 Jones Bldg., Tacoma 2. Secretary, Eugene E. LaCroix, 1747 S. Sheridan Ave., Tacoma 5.

State and National Boards

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations September 20 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations September 7-8 at the Y.M.C.A. Bldg., E. 16th Ave. and Lincoln St., Denver. Applications must be filed on or before August 24. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations October 8 in Room 23, Lampson Hall, Yale University, New Haven. Applications must be filed 2 weeks prior to examination. Address Miss M. G. Reynolds, executive assistant, Board of Healing Arts, 52 Whitney Ave., New Haven.

DISTRICT OF COLUMBIA

Basic science examinations in October. Address Mr. Paul Foley, Deputy Director, Department of Occupations and Professions, 1740 Massachusetts Ave. N.W., Washington 6.

HAWAII

Examination dates by Territorial Law are usually the first Wednesday, Thursday, and Friday of January, April, July, and October. One of these dates is set for examination after approval of candidate's application by the Board. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Kauikeolani Bldg., Honolulu 13.

ILLINOIS

Examinations in October in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations October 11 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

MARYLAND

Examinations in October. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 No. Charles St., Baltimore 1.

MICHIGAN

Basic science examinations in October. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Bldg., Lansing.

MINNESOTA

Basic science examinations October 4-5 at the University of Minnesota, Minneapolis. Applications must be filed by September 10. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

MONTANA

Examinations September 6. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

Dr. Willard has been reappointed to the Board for a term expiring May 13, 1959.

NEBRASKA

Basic science examinations October 4-5. Applications must be filed 15 days prior to examination. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

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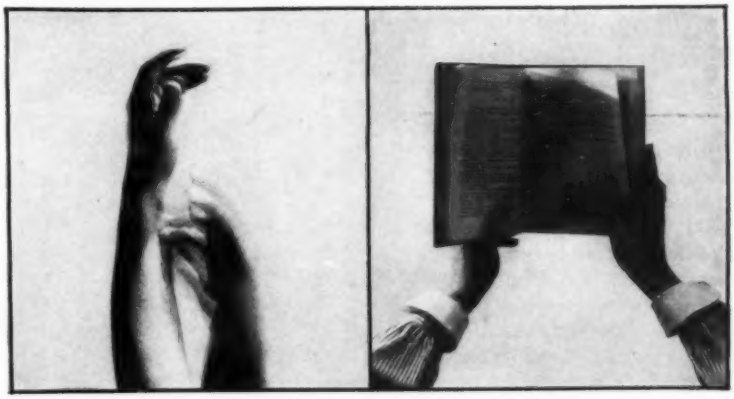

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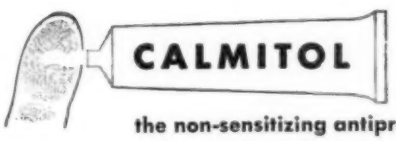

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1. Lubowe, I. I.: New York State J. Med. 50:1743, 1950.

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NEVADA

Basic science examinations October 4. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations September 14-15 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations July 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P. O. Box 1522, Santa Fe.

NEW YORK

Examinations October 4-7. Address John W. Paige, M.D., chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

OREGON

Basic science examinations September 10. Address Mr. John R. Richards, secretary, State Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations October 6-7. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations August 31

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1. Overall, J. C.: Southern M. J. 47:789, 1954. 2. Editorial: New England J. M. 246:111, 1952. 3. Grayzel, H. G., Heimer, C. B., and Grayzel R. W.: New York St. J. M. 53:2233, 1953. 4. Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951. 5. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949. 6. Turell, R.: New York St. J. M. 50:2282, 1950.

in Room 366, State Office Bldg., Providence. Applications must be filed approximately 20 days prior to examination. Address Mr. Casey.

SOUTH DAKOTA

Professional examinations July 19-20 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

WISCONSIN

Basic science examinations September 23 at the Assembly Chamber, State Capitol, Madison. Applications must be filed by September 15. Address Mr. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

WYOMING

Examinations October 3 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

Within 60 days after July 1—Indiana, $5.00 for residents, $10.00 for nonresidents. Address Paul R. Tindall, M.D., secretary, Board of Medical Registration and Examination, 538 K. of P. Bldg., Indianapolis 4.

September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, 21 W. Broad St., Columbus 15.

During September—Nebraska, $2.00. Address C. Eugene Brown, D.O., secretary, Osteopathic Board of Examiners, 1702 Second Ave., Nebraska City.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles; therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic As-

sociation for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

EFFECT OF GAMMA GLOBULIN ON MEASLES*

By Otto L. Bettag, M.D., Frederick Plotke, M.D., and Harold M. Sterling, M.D.†

The incidence of complications with or following measles has led to various attempts at modification or prevention (*1, 2*). At present, the use of gamma globulin is considered the most effective means of preventing measles (*3, 4*). Since it is important in some instances to prevent this illness and in others to modify it, much effort has been directed recently toward establishing an optimum dosage (*5, 6*).

Current recommendations call for the administration of gamma globulin by the intramuscular route in a dosage of approximately 0.02 cc. per pound of body weight for modification and of 0.1 cc. per pound of body weight for prevention of measles (*7-9*). At least one worker has presented evidence that 0.02 cc. per pound of body weight is excessive since this "modifying" dosage frequently appears to prevent the disease completely (*10*).

The effectiveness of gamma globulin in preventing or modifying the course of measles was studied during an epidemic in the spring of 1954 at the Illinois Children's Hospital School, Chicago.

The study included 92 permanent resident children at this institution. At the time of this report, the shortest period of residency in this hospital school was 2 and the longest 8 years.

The diagnostic categories included 35 children with cerebral palsy, 26 children with residual paralysis following poliomyelitis, 5 children with paraplegia following trauma, 2 children with paraplegia following transverse myelitis, 3 with paraplegia due to spina bifida, 5 with muscular dystrophy, and 16 with other disorders. The children in general represented the more severe forms of these conditions. Approximately one-third of the students are almost or totally helpless. In this latter group illness of any kind or complications thereof would be serious.

The review of the status of the children at the time of this study showed that of the 92 children, 16 had no history of exanthemata; 66 had a definite history, and 10 had a questionable history of measles.

The age range for the group was 5 to 20 years. The average age was 12 years; 82 were white, and 10 were Negroes; 52 were males and 40 females. The patients were observed under the condition of this study from March 1, 1954, to May 15, 1954.

For the study the children were divided into three groups:

Group 1 comprised 58 children in good physical condition who had a positive history of measles. These children were considered immune, and gamma globulin was not administered to them (*11*).

Group 2 consisted of 24 children in good physical condition who by history were considered susceptible. "Modifying" doses of gamma globulin were given to this group (*12*).

*Reprinted from *Public Health Reports*, April, 1955.

†Dr. Bettag is director of the Illinois Department of Public Welfare, and Dr. Plotke is chief of the department's public health service. Dr. Sterling is medical director of the Illinois Children's Hospital School, Chicago.

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Reference to RHINALGAN:
1. Van Alyea, O. E., and Donnelly, W. A.: E.E.N.&T. Monthly, 31, Nov. 1952.
2. Fox, S. L.: AMA Arch. Otolaryn., 53, 607-609, 1951.
3. Molomut, N., and Harber, A.: N.Y. Phys., 34, 14-18, 1950.
4. Lett, J. E., (Lt. Col. MC-USAF) Research Report, Dept. Otolaryn., USAF School Aviat. Med., 1952.
5. Hamilton, W. F., and Turnbull, F. M.: J. Amer. Pharm. Ass'n., 7, 378-382, 1950.
6. Browd, Victor L.: Rehabilitation of Hearing, 1950.
7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

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Group 3 included 10 children for whom measles, or its complications, was considered dangerous. This group was protected by the administration of "preventive" doses of gamma globulin, irrespective of a previous history of measles.

Group 3 consisted of:

Two students with previous history of measles, both manifesting severe respiratory difficulties following paralytic poliomyelitis.

Two students with indefinite history of measles; both had progressive central nervous system degenerative disease.

Six students with no history of measles—3 severe dystonic athetoid cerebral palsy cases, 2 muscular dystrophy cases, and 1 spastic paraplegia case with primary tuberculosis infection.

The first case of measles in this epidemic developed March 5, 1954, in a child who was isolated in his room from the onset of the symptoms (except for part of one day) 3 days prior to the appearance of a rash. It was later learned that during a visit with relatives he was in contact with a child who subsequently developed measles. Immediately after the rash appeared in this first case, the child's entire residential floor was quarantined in an attempt to prevent the spread of the disease. Gamma globulin was administered to all those for whom it was indicated on the fourth day of probable exposure.

During the following week several new measles cases appeared, but none occurred on the residential floor originally quarantined. As soon as the new cases were diagnosed, the 24 students in group 2 presumed susceptible because of no known previous attacks of measles were moved to a single residential floor. The group 1 students with natural immunity and the group 3 children with immunity passively conferred by gamma globulin continued as usual their activities of school, treatment, and home visits. For those quarantined, schooling and the various types of treatment were carried on in their quarters.

Six of the ten students in group 3 subsequently developed measles. One had an onset 11 days, two 12 days, one 17, one 18, and one 38 days after gamma globulin administration. Four children had mild and 2 had moderate cases. There were no significant sequelae to the infection. Thus, of the 10 persons for whom measles was presumed to be dangerous, 6 contracted the disease in spite of administration of the accepted "protective" dose.

The 2 children afflicted with moderate cases of measles also developed German measles 30 and 45 days, respectively, after the onset of their measles. Two others who did not have measles developed German measles approximately 6 weeks after gamma globulin administration. One of these students, with progressive central nervous system degeneration (Pelizaeus-Merzbacher syndrome), appeared to deteriorate rapidly following relatively mild German measles.

A breakdown of patients developing measles after a "protective" dose of gamma globulin given at least 11 days prior to onset of symptoms was made. The gamma globulin failures were distributed among a wide range of degenerative diseases.

Of the 24 students who received "modifying" doses of gamma globulin, 19 developed measles, and in 8 of these German measles developed subsequently. Four of the 19 had severe cases of measles and 4 others had complications of some sort.

Of the 58 students with positive histories of measles, 20 students developed measles, 4 German measles, and 3 had both. Three had some type of complications.

SUMMARY AND CONCLUSIONS

An epidemic of measles followed by an epidemic of German measles occurred in a residence school for 92 orthopedically disabled children, 16 having no previous history of exanthemata. The possibility of progression of basic conditions or of seriousness of sequelae prompted an attempt to prevent the disease in 10 pupils by the administration of 0.1 cc. gamma globulin per pound of body weight intramuscularly on the fourth day after exposure; 24 others received 0.02 cc. per pound of body weight to modify the illness; 58 students, who had positive histories of measles, received no gamma globulin and, therefore, were not quarantined.

Six children developed measles at least 11 days after receiving a "preventive" dose of gamma globulin. Of these, 4

had mild and 2 had moderate measles, but none had complications from this disease. One child suffered rapid progression of the basic condition following a subsequent attack of German measles.

A previous history of measles was of no value in deciding who should receive gamma globulin, since 20 students with such histories developed measles, 4 German measles, and 3 both. Three had complications of some type.

Gamma globulin in the dosage currently recommended for prevention of measles failed to protect 6 out of 10 children to whom it was administered. Gamma globulin in "modifying" dosage apparently failed to modify the disease in 4 out of 19 students to whom this dosage was administered. There was no evidence that gamma globulin prevented or modified German measles.

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(2) Sweet, C.: Measles: Its prophylactic treatment with blood of immune persons. California & West. Med. 38:254-260 (1933).

(3) Ordman, C. W., Jennings, C. G., Jr., and Janeway, C. A.: Use of concentrated human serum gamma globulin in prevention and attenuation of measles. J. Clin. Investigation 23:541 (1944).

(4) Janeway, C. A.: Clinical use of products of human plasma fractionation. J. A. M. A. 126:674-680 (1944).

(5) Greenberg, M., Frank, S., and Rutstein, D. D.: Gamma globulin and placental globulin. J. A. M. A. 126:944-947 (1944).

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(8) Coigny, R. L., and Straus, P.: Epidemiological study of measles as influenced by mass immunizations. Presse Méd. 59:634 (1951).

(9) Christensen, P. E., and Schmidt, H.: An epidemic of measles in southern Greenland, 1951. IV. Significance of specific prophylaxis. Acta med. Scandinav. 145:126-142 (1953).

(10) Bivings, L.: Gamma globulin dosage and measles control. J. Pediat. 43:401-403 (1953).

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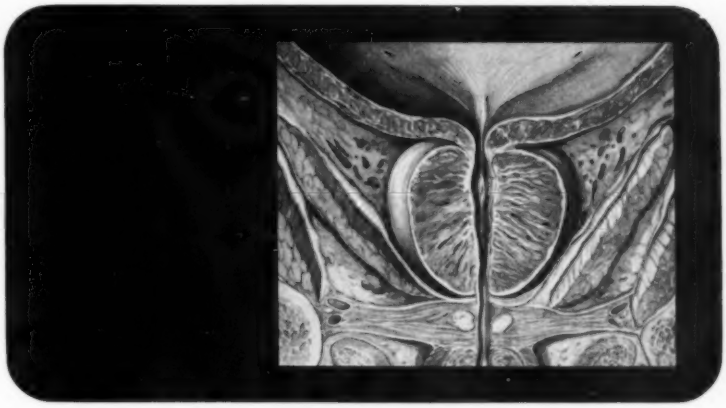

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GONORRHEA DETECTION BY URINE EXAMINATION*

By S. Ross Taggart, M.D.†

An intensive epidemiological and public educational program directed toward gonorrhea control has been disappointing because it has not been successful in rapidly reducing the number of cases of gonorrhea being reported. If control is to be accelerated, additional case-finding measures must be established.

In hospitals, clinics, and physicians' offices, urine specimens of large numbers of patients are routinely examined for albumin, sugar, and so on. If, at this same time, gonorrhea could be found by examination of these urine specimens, an economical and simple case-finding aid would be available; a rapid screening procedure for detection of gonorrhea suspects would be provided. This paper presents the results obtained in a preliminary study of macroscopic inspection and microscopic examination of the sediment of urine specimens for the detection of gonorrhea in patients from a selected clinic group and from an unselected screening line group.

METHODS

Urine specimens were collected in flat-bottomed specimen bottles. One to two cubic centimeters of 10-percent acetic acid was added to the fresh urine specimens, which were then macroscopically inspected. An opinion was recorded as to the presumptive presence of pus or shreds in the urine specimen. If shreds or pus were present, the specimen was considered presumptively positive for gonorrhea.

The urine specimen was then allowed to sit for at least 1 hour. After this period it was decanted and a spread was prepared from the sediment. The spread was air-dried, stained with a Gram's stain, and examined under an oil-immer-

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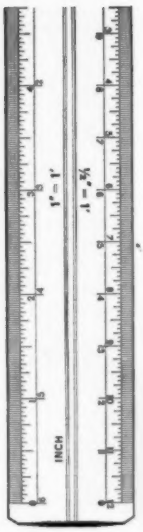
†Dr. Taggart is acting chief, preventable and chronic diseases division, bureau of disease control, District of Columbia Department of Public Health, Washington, D. C. This paper was presented at the Symposium on Recent Advances in the Study of Venereal Diseases, April 29-30, 1954.



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sion lens for the presence of gram-negative intracellular diplococci. Urine specimens which had been allowed to stand for any period before initial visual inspection were swirled before acidifying and then were allowed to stand for at least an hour before microscopic examination of the sediment.

When a microscopically positive sediment was found in a urine specimen and a diagnosis of gonorrhea had not been established in the patient prior to the urine test, the patient was re-examined bacteriologically. The diagnosis of gonorrhea was established or confirmed in all patients with positive sediment findings by urethral or cervical spread or, if indicated, by culture.

RESULTS

The selected population group consisted of male patients who were examined in a venereal disease clinic. Of this group, 68 had a frank urethral discharge in which gonococci could be demonstrated. Using the test technique described in this paper, 52 of these 68 patients, or approximately 75 percent, were found to have gram-negative intracellular cocci in the sediment of their urine specimens; these urine specimens also had been considered positive for gonorrhea by sample inspection, utilizing the technique described for screening. Thirty-two of these venereal disease clinic patients had no urethral discharge and had been considered clinically negative both by history and physical examination. However, in 3 of the patients (approximately 10 percent), positive findings in the sediment were obtained by microscopic examination (2 were positive and 1 was negative by macroscopic inspection). These diagnoses of gonorrhea were confirmed by bacteriological study of urethral scrapings.

In order to determine the effectiveness of this simple macroscopic technique under conditions in which patients were not appearing primarily for venereal disease diagnosis and treatment, it was applied to a different type of patient body. This group consisted of patients of the outpatient service of the District of Columbia General Hospital. Urine specimens from these patients are collected routinely as a part of their initial examination. These patients were being seen for various medical and surgical complaints, and without this examination would have been considered free of gonorrhea. The urine specimens were examined by the test technique under consideration and the discovery rate of gonorrhea in this group was 15.0 percent in the males and 6.8 percent in the females. Demonstration of gonococci by urethral or cervical spread or culture was possible in all of these patients. Because this study was of a preliminary investigative nature, sugar fermentation studies for definitive bacteriological diagnoses were not considered necessary at this time.

DISCUSSION

The results of this study of the detection of gonorrhea suspects by macroscopic examination of the sediment of urine specimens appear encouraging, and suggest the desirability of further study to determine whether this method might become practicable.

It would appear that a technique of macroscopic inspection followed by microscopic examination of sediment of presumptively positive urine specimens will establish a diagnosis of gonorrhea in approximately three-fourths of an infected group.

Culturing the urinary sediment of all patients would no doubt have raised the percentage of infected persons found. However, the technical difficulties and increased cost entailed by this procedure would, in most cases, not be warranted. Of greater significance to the gonorrhea control program is the fact that a simple method of screening large numbers of individuals who are infected with gonorrhea, but who have not been motivated to seek specific medical care, should be of value in the public health control of gonorrhea. From experimental inoculation studies with gonorrhea it is known that not all patients have the profuse outpouring of pus and urethral discomfort that are considered characteristic of the disease. It is this group, not sufficiently disturbed by the infection to seek specific care, which might be discovered, especially if the portion of the population in which the prevalence of gonor-

rhea might be expected to be high were screened.

The finding of an infection rate of 15 percent among a clinic group consisting of patients who were not destined for a type of examination which could be expected to bring them to treatment for gonorrhea suggests the potential importance of this simple method of urine examination. It would appear that the preliminary studies herein described are sufficiently valuable to suggest the desirability of further investigation. It is known that gonorrhea thus far has been relatively resistant to various control procedures. In spite of simple, sure cure the disease has not responded to our control efforts in the same way as syphilis. One of the stumbling blocks has been the lack of a simple case-finding device. These preliminary results would seem to merit further investigation.

SUMMARY

A simple, routine screening procedure for a presumptive diagnosis of gonorrhea by a macroscopic examination of urine samples has been described. In a high-prevalence population, such a technique should be an aid in case finding in a gonorrhea control program.

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THE OUTLOOK FOR MENTAL PATIENTS*

Mental disorders constitute a large and growing health problem in the United States. In the past 20 years, the number of first admissions to hospitals for the long-term care of psychiatric patients has increased more than 60 percent and the number of patients under care in these institutions has risen almost as rapidly. Currently, nearly two fifths† of the total bed capacity of the hospitals in our country is devoted to the care of mental patients. Even so, a great many people with mental disorders are unable to obtain the benefits of institutional care because of the shortage of personnel and facilities in mental hospitals.

Although the problem of mental disorders is a very serious one, certain features of it appear worse than they really are. For example, a substantial part of the rise in hospitalization for mental disorders reflects merely the increase in population and the growing proportion of people at the older ages. In New York State, about 30 percent of the increase in first admissions of women to mental hospitals over the last two decades is attributable to these population factors. It is also significant that patients with mental disorders due to conditions associated with old age account for an increasing proportion of all first admissions to mental hospitals; such conditions now constitute one fourth of the total first admissions.

A more enlightened attitude has lessened the reluctance of the public to seek institutional care for persons with mental disorders. Because the proportion of people living in small urban dwelling units has increased, mental patients who in the past might have been kept at home are now more likely to be placed in hospitals for treatment. Still another factor contributing to the rise in hospitalized patients is their increasing length of life. The longevity of mental patients, as of the population as a whole, has improved as a result of the control gained over the infectious and other diseases.

Substantial progress has been made in the treatment of various types of mental disease. Shock therapy in various forms has been reported as helpful in an increasing number of selected cases. New surgical techniques and recent developments in drug therapy also hold out promise of restoring some patients to useful life. Other new therapeutic techniques, growing knowledge of mental disorders, and improvement in institutional management have also contributed

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, April, 1955.

†Exclusive of beds in registered hospitals for epileptics and mental defectives which account for another tenth of the total.

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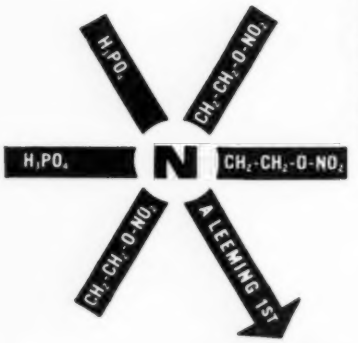

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toward raising the proportion of recoveries among mental patients.

The long-term outlook after recovery is likewise favorable. A recent study covering the experience of a large number of Life insurance companies during the period 1935-1950 showed that applicants with a history of psychoneurosis of relatively mild degree (most of whom were men so diagnosed in connection with military service) had a survivorship rate of 99.1 percent five years after they were insured and 97.3 percent ten years after being insured, or only slightly less than the corresponding proportions for standard risks as a whole. For those with moderate illnesses of this kind, the survivorship record after recovery was not quite as good.

Even for applicants with a history of mental illness of severe degree, who naturally were selected with great care, survivorship after recovery compared not too unfavorably with standard risks. Thus, at the end of 15 years the proportion surviving among those with severe psychoneurosis or psychosis was 88.8 percent compared with 93.1 percent among standard risks as a whole. The results indicated that the excess mortality among patients with a history of mental disorders is no greater than that resulting from many types of physical impairments. Suicide accounted for a large part of the excess mortality among them. For most other causes of death, their mortality was not significantly different from that among standard risks.

A follow-up study‡ of a representative sample of World War II veterans who had suffered psychoneurotic breakdown during their military service also showed generally favorable results with respect to their condition at time of follow-up about five years after their breakdown. In this study most of the veterans were interviewed by psychiatrists at follow-up. It was found that 85 percent of the men were gainfully employed, and all but a small proportion of these were in full-time employment. Two thirds of them had had no psychiatric treatment after discharge from the service, and in only 2 percent had there been intensive or prolonged treatment by psychiatrists. Less than 10 percent showed evidence at follow-up of serious psychiatric disability.

Fortunately, the attack on the problem of mental illness has been gaining momentum in recent years. More and more physicians are selecting the field of psychiatry for special training, while greater attention is being given in medical education to the importance of recognizing and treating mental and emotional disorders. Various measures are being taken to increase both the number and qualifications of professional and custodial personnel in hospitals. Voluntary and official agencies are making vigorous efforts to alert the public to seek early treatment for mental and emotional disorders, and to gain wide public support for the mental health program. Research is yielding new knowledge on the prevention, diagnosis, and treatment of these disorders. While much is being done, the effort is still far from commensurate with the magnitude of the problem. There is good reason to expect that an intensified and broadened program would bring marked progress in the field of mental health as it has in so many others.

THE BABY BOOM CONTINUES*

The baby boom which started at the close of World War II continues unabated. From 1946 through 1954 the number of births in the United States averaged in excess of 3¾ million annually, or 1-3/5 times the number in 1933. Births reached an all-time high of 4,076,000 in 1954, and the end of the boom may still be several years ahead.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, April, 1955.

‡Norman O. Brill and Gilbert W. Beebe, "Follow-up Study of Psychoneuroses," *The American Journal of Psychiatry*, Vol. 108, No. 6, December 1951, p. 417.

An important factor in the recent upsurge in births has been the marked rise in the number of married women. Wives at ages 15-44 have increased in number by one seventh since 1945, and by more than one third since 1933. A second factor has been the almost uninterrupted rise in the fertility rate from its low level in the 1930's. In each of the postwar years about one out of every six married women aged 15-44 bore a child, but in the mid-1930's the proportion was only one in eight.

The baby boom started with a sharp upswing in first births, their number increasing by almost two thirds in the two years from 1945 to 1947. This sharp rise reflected not only the high marriage rate following demobilization, but also the return of many servicemen to family life. At the peak in 1947, first children were born at the rate of 69 per 1,000 married women aged 15-44. Subsequently, however, the rate declined rapidly, parallel to the general decrease in marriages. By 1949, it had fallen to 52 per 1,000; currently the rate is about 46 per 1,000. Since no pronounced rise in marriages is expected before the next decade, the rate for first births may fall somewhat further before it again swings upward.

Particularly noteworthy has been the sustained high rate for second births, which for nine consecutive years has continued at the highest level in at least a generation. In this period, the rate for second births averaged close to 45 per 1,000, compared with rates below 30 in the years 1933-1937. Moreover, since 1951 there have been somewhat more than 1,100,000 second births annually, which is just short of the number of first births—a situation without precedent since birth records have been maintained in our country.

Equally striking has been the rapid rise in the rate for third births. From its low point of 17 per 1,000 in the late 1930's, the rate for third births has climbed to 31 per 1,000—the highest in more than a third of a century. The birth rate has also been on the increase in larger families, and the indications are that it will continue upward for a while longer. The rate for fourth children, for example, has increased from its low point of 10 per 1,000 in 1939-1942 to 18 per 1,000 in 1954. The rate for fifth births is now also showing an upward tendency. Nevertheless, in 1954 it was still only 10 per 1,000, which is at the level of the rate in 1929, but well below that for earlier years.

With the continuation of favorable economic and social conditions, the expected decline in the birth of second and third children may be offset in considerable degree by the rise in births of fourth and higher orders. The outlook is that the total number of births will decline only gradually during the balance of this decade.

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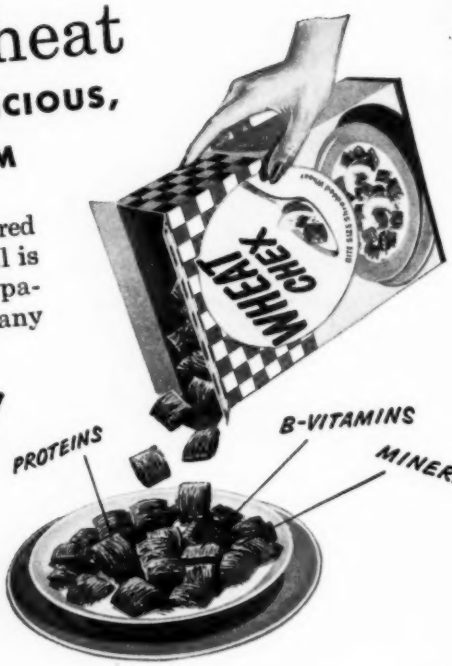

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Moreover, the effect of a single application *lasts* for 6 to 10 hours or more, permitting uninterrupted sleep throughout the night.

Colorless, greaseless and nonstaining, EURAX is invisible following application—especially important when "summer pruritus" affects exposed parts of the body.

EURAX® (brand of crotamiton) Lotion is available in 2 oz. prescription bottles, and larger size dispensing bottles. Also available—EURAX Cream in tubes of 20 Gm. and 60 Gm., and 1 lb. dispensing jars.

Geigy

GEIGY PHARMACEUTICALS

Division of Geigy Chemical Corporation, 220 Church Street, New York 13, N.Y. *In Canada:* Geigy Pharmaceuticals, Montreal

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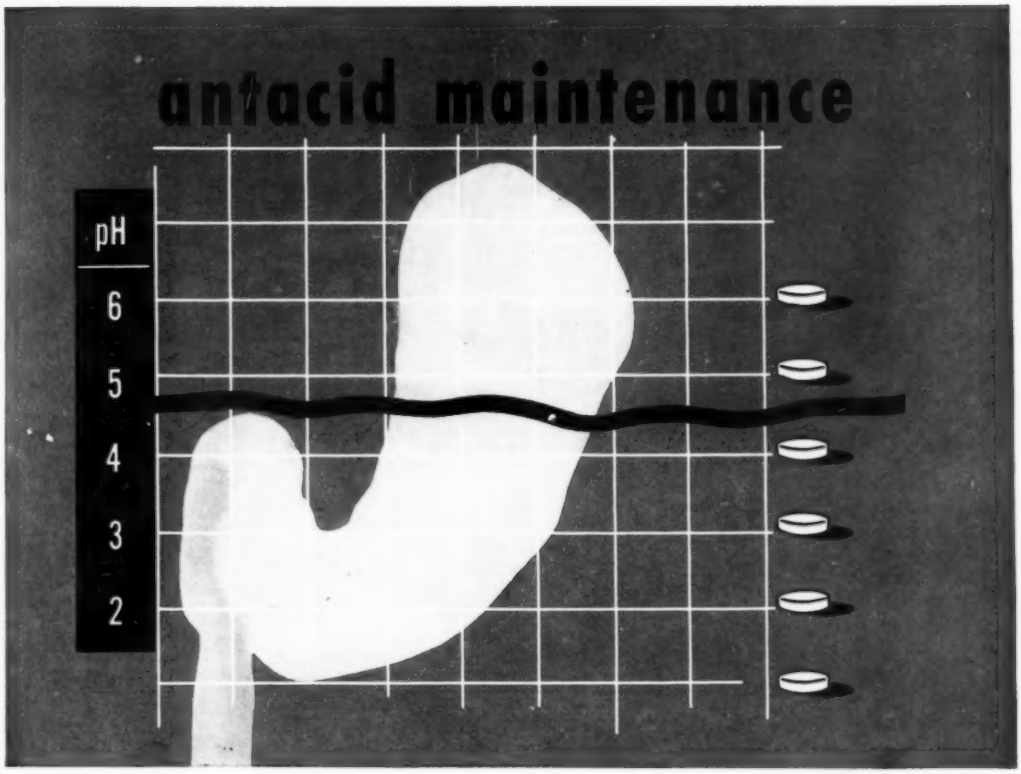

Healing of peptic ulcer must be followed by effective antacid maintenance therapy to prevent recurrence. This can be achieved conveniently with agreeable, easy-to-carry Creamalin Tablets and Capsules.

Through sustained reduction of gastric acidity without the danger of alkalosis, nonabsorbable Creamalin provides reliable and safe antacid control for the ambulatory ulcer patient.

REACTIVE ALUMINUM HYDROXIDE GEL

Creamalin®

TABLETS: Bottles of 50 and 200
CAPSULES: Bottles of 100
LIQUID: Bottles of 8 and 16 fl. oz.

Winthrop-Stearns INC.
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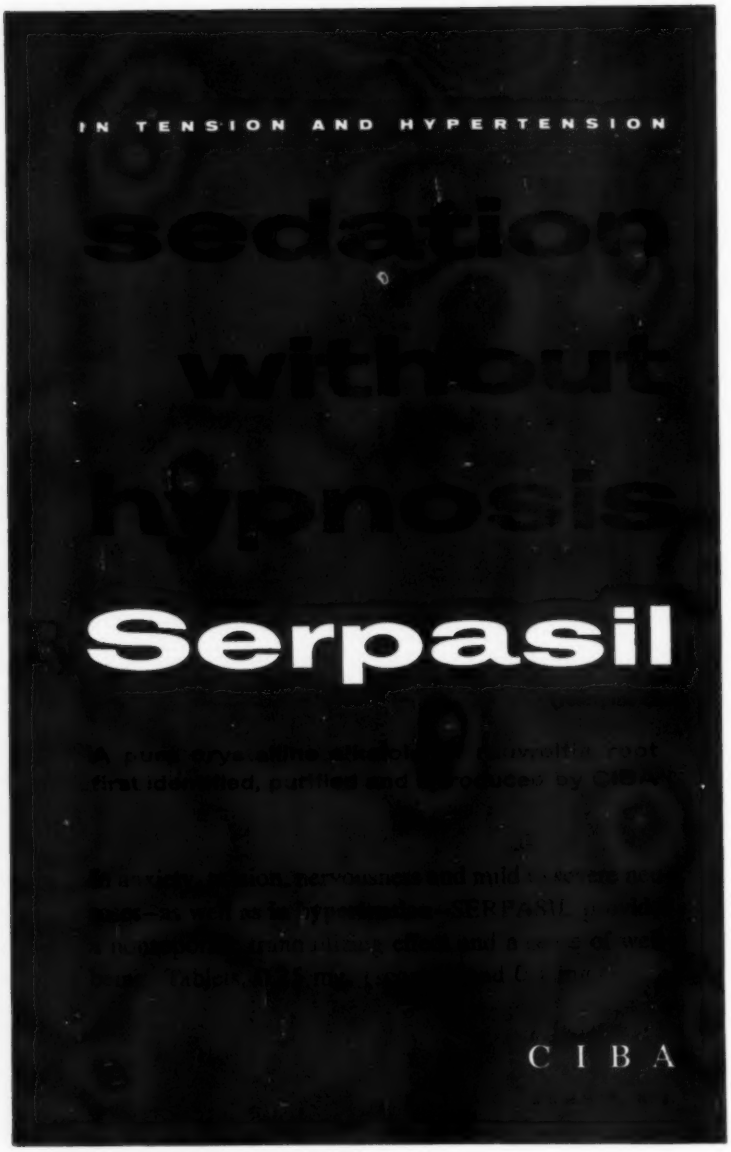
CREAMALIN, trademark reg. U. S. Pat. Off.



New! SERPASIL® ELIXIR
Each 4-ml. teaspoonful contains 0.2 mg. of Serpasil